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SYMPOSIUM ON UPPER RESPIRATORY TRACT AND SOME OF THEIR COMPLICATIONS.

I.—THE RELATIONSHIP BETWEEN INFECTIONS OF THE UPPER RESPIRATORY TRACT AND THE EAR.*

DR. JAMES G. DWYER, New York.

This is a very broad subject and hours could be allotted to the consideration of these infections and their manifold manifestations. In our part of the symposium, we wish to stress some of the important old facts and call attention to some of the newer developments of recent years.

It might be said, broadly speaking, that the great majority of the purulent lesions of the ear are directly or indirectly secondary to conditions in the upper respiratory tract. From the connection between the nasopharynx and the ear through the Eustachian tube and through the circulation, both blood and lymph and also the sinuses, especially the sphenoid, and the ear, the latter is always potentially involved. This does not mean that the infection travels only through the Eustachian tube, but by other paths which will be considered later. Another point to be stressed is the important role that certain vitamins play in the nutrition of the nose, throat, sinuses and teeth and whose absence or deficiency affect the nose and throat and thus, indirectly, the ear. This is true also with

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regard to the endocrines and in the allergic phenomena and especially true in the upper respiratory tract in the conditions found especially in children in acidosis. Several of these points will be referred to later.

Limiting the discussion in this paper to the upper respiratory tract and its infections, we would say that most of these infections reaching the ear from this area do so by way of the nasopharynx, Eustachian tube, or through the vascular or lymph stream or both. The channel along which morbid processes most frequently reach the ear is the Eustachian tube, especially in early life, when the tube occupies a more horizontal position than in the adult and is also relatively shorter, straighter and wider. At this period of life it is also more in relationship with the digestive tract and is consequently more liable to contaminating influences. From the histological standpoint, we must also remember that the layer of ciliated epithelium in the cartilaginous portion of the tube lies directly upon a layer of adenoid tissue of variable thickness. This adenoid stratum has been called the tubular tonsil. In the young child, the adenoid tissue of the tube is more developed than in the adult and assumes the form of prominent lymph follicles: hence occlusion of the tube occurs much more frequently in childhood than in later life, because of the swelling of this tissue. The mucus glands are acinous in structure and form a thick layer, frequently interrupted by a stratum of fibrous tissue. Isolated glandular ducts occur throughout the adenoid tissue. Both the mucus glands and the adenoid tissue decrease towards the isthmus of the tube.

From the foregoing then, it is easy to see that a very frequent and prolific source of morbid affections of the tubotympanic axis is disease of the nasal and nasopharyngeal mucosa, such as rhinitis, nasopharyngitis, acute or chronic postnasal adenoids and infections of the nasal accessory sinuses. Measles, scarlet fever and diphtheria have frequently a most injurious influence upon the ear; the inflammatory process, often exceedingly violent, spreading up the Eustachian tube, and ending in destruction of the whole or part of the contents of the tympanic cavity. Other catarrhal affections of the respiratory tract, such as laryngitis, bronchitis and pertussis, are also prone to be followed by involvement of the ear, as the result either of direct continuity of tissue or from secondary microbic invasion. Of course, we all know that the

injudicious or unskillful use of the nasal douche or nasal instruments is also at times followed by reaction phenomena.

One of the conditions met with frequently in children is that of the acute catarrhal ear or O.M.C.A. These attacks are usually due to an altered condition of the nasopharyngeal axis, with more or less interference with the Eustachian tube, and thus the production of an acute nonpurulent inflammation of the middle ear. The pathological findings vary from a retracted drum, due to the negative pressure, to one in which there is a nonpurulent exudate in the middle ear. This exudate may be as clear as water and rapidly absorb on the removal of the cause. On the other hand, it may be quite cellular and fibrinous and lead to permanent changes in the middle ear and thus to deafness. Add to this the fact that such an attack may last for weeks or months without pain, and that with every cold or congestion, this may recur over years, the serious nature of this lesion is apparent. It undoubtedly is the cause of deafness in adult life in a certain percentage of the cases, but the writer has never accepted this as the cause of the vast number of cases of O.M.C.A. Some are of focal infection origin; others are due to faulty metabolism; others are due to avitaminosis. The most of them are unexplained, as far as our present knowledge goes.

Acute Infections: The next class of ear diseases comprise the infections of the middle ear, mastoid, etc., with which we are working every day, and which are probably the best understood part of our work. Most infections probably travel through the Eustachian tube, but one of the points the writer wishes to stress is that quite a larger percentage than we think travel through the blood or the lymphatics and produce quite a different pathology. Thus instead of a mastoid through the usual path of tube, tympanum and mastoid process, we may get an invasion from the sphenoid or from tissue around the Eustachian tube and thus get an osteomyelitis of the base of the skull and only secondarily an invasion of the mastoid process proper. This process may occur as a thrombophlebitis, leading to an osteomyelitis. This may be the pathway in supuration of the petrous bone and not from the mastoid process proper. The importance of this observation cannot be stressed too much. To our mind it explains some of the fatal cases of so-called mastoiditis, operated upon as an ordinary mastoiditis, but which is essentially an osteomyelitis of the base of the

skull, or the petrous bone, in the mastoid region, and usually fatal. It also explains to our minds some of the differences of opinion among our otologists, in reference to the pathology of the petrous bone. Some claim that we have to have a pneumatic mastoid, with cells leading to the petrous tip in order to produce a lesion of the petrous. Others claim that it is an osteomyelitic process and that the path is not necessarily from an infection in the mastoid. We have performed autopsies in two cases of meningitis in which we could demonstrate that it was undoubtedly an osteomyelitic process, and we are convinced that these patients were doomed from the start, as such lesions along the base are not amenable to surgery in this region and tend to go on to a fatal issue.

At this point, it is interesting to consider the bacteriology of acute mastoiditis. Some years ago, we ran a series of hundreds of consecutive mastoids, from which we cultured the pus from the operative mastoid on the table. The findings were as follows: Ninety-one per cent were streptococcus hemolyticus. Seven per cent were the various strains of the pneumococci, and the rest staphylococci with an occasional other organism. We think, however, that these proportions would vary with changes in the season and with epidemics affecting the nose and throat.

Other classes of infections are those known as the focal infections, in which the teeth, tonsils or nasal sinuses may indirectly cause a neuritis of the auditory nerve, with noises and deafness, or an irritation of the labyrinth, with vertigo and dizziness, or in some of the O.M.C.C. cases, where we get a low grade inflammation of the joints of the middle ear. How large this class of cases is the writer does not know, but he has seen quite a considerable number.

There are three conditions that profoundly affect the upper respiratory tract both directly and indirectly, and also the ear. These three are respectively avitaminosis, endocrine disorders and allergic phenomena. Unfortunately, none of these conditions have been working out sufficiently for us to be sure of our ground in any given case, but we will all be doing better work on otology and rhinology, as our knowledge of these three conditions increases. We know that certain vitamins affect the pulp of teeth; others the epithelium of the upper respiratory tract, etc. We also realize the difficulties in dealing with vasomotor conditions.

300 Park Avenue.

SYMPOSIUM ON UPPER RESPIRATORY TRACT AND SOME OF THEIR COMPLICATIONS.

II. — UPPER RESPIRATORY INFECTIONS FROM THE PEDIATRIC STANDPOINT.*

DR. LOUIS C. SCHROEDER, New York.

Any one of a dozen problems, bearing upon the upper respiratory infections is deserving of the time so graciously given by your section to a pediatricist. Of a truth, there is much in common between us and it is an embarrassment of riches which makes it difficult to choose just what will be of the greatest mutual interest.

It has seemed wiser to forego any search of the literature or the presentation of cases and to adopt a somewhat reflective turn of mind in giving the present pediatric viewpoint. After all, pediatric literature is filled with the experience of pediatricists. It is quite certain that this sum total easily surpasses the experience of a single individual no matter how wide his experience or successful his therapeutic measures.

There is little need of dilating upon the importance of the subject. There is need, however, of reminding ourselves from time to time of it. So often, in the desire to get therapeutic results, the great opportunity of preventing recurrences, a side of preventive medicine so seldom thought of, is overlooked. How common for instance, is the knowledge that 2,000,000 children in our country have impaired hearing; that almost 500,000 have definite cardiac limitations and that there are 400,000 tuberculous children? Here alone are nearly 3,000,000 children, the condition of whose upper respiratory tract may have been the factor which threw the balance against them. If there are those who will quibble because the tuberculous children are included one can easily substitute a large number of children who have chronic lung afflictions of a nontuberculous origin. Add to these the variegated cases of focal infections, the chronic arthritides and the impaired renal function cases from infected upper respiratory tracts and one

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can visualize an army of handicapped children the size of which is staggering. The preventive pediatric standpoint to which all far seeing otolaryngologists subscribe is that the upper respiratory infections and reinfections must be prevented and as the White House Conference succinctly added, "Those already handicapped must not be dismissed as liabilities but considered as social assets."

As inviting as a further perusal of methods looking toward prevention is, it seems wiser to think about problems more directly concerned from your angle and ours and trust as these are discussed that the best methods of prevention will present themselves.

To those of us, whose days in medicine stretch back far enough into the past, to have been witnesses of the old process of decapitating tonsils when the clinics resembled the Place de la Concorde with the students taking the places of the knitting women, the present clean, practically bloodless dissections represent an advance of far reaching and major importance. How many other developments have there been which are of as much significance? Can it be said that all physicians are in possession of knowledge which will enable them to make a correct decision as to whether tonsils should be removed? As a certain ex-governor of our State usually remarks, "Let us look at the record."

Let us look at a rather remarkable survey of health procedures in our public schools, recently published by the American Child Health Association. In the chapter on School Health Influence on Tonsillectomy there are certain facts and conclusions which make one pause.

A sampling of 1,000 children 11 years of age, were taken and 61 per cent were found to have had their tonsils removed. Those with tonsils were then examined by a group of physicians and 45 per cent of these were recommended to have their tonsils out. Those remaining from this examination were shortly afterward examined by another group of physicians and 46 per cent of these were recommended for tonsillectomy. Those still with tonsils were then examined and 44 per cent of them were recommended for tonsillectomy. The following conclusion is worth thinking about. The high average proportion for tonsil removal made by each of the groups of physicians when examining cases that had been passed as not need-

ing any corrective attention at a previous examination, emphasizes the extreme lack of agreement among physicians and indicates a strong tendency to lean toward tonsillectomy.

This confusion worse confounded, must be a matter of regret to all of us. It will not do to say that such a series of events would not happen to pediatricists or laryngologists because, combined, they care for an infinitely smaller proportion of the child population than does the general practitioner.

The practical problem is to determine the indications for tonsillectomies, giving far more weight to the history than is usually accorded. To the suggestion that an experiment in a limited district be undertaken, with the object of working out a more satisfactory technique, most pediatricists are in full accord and they feel that laryngologists should be pioneers in setting such a study in motion.

Our lack of knowledge of the physiology of the tonsil is abysmal. With the exception of one or two, it is known that all mammals have them but, beyond the fact that they open into the pharynx there is very little agreement as to their function. Most pediatricists feel that the secret of the prevention of the upper respiratory infections will only be known when the nature of one or more viruses or micro-organisms is definitely understood and when some clearer understanding of that elusive function of the human frame which is called "resistance," is available. Having this belief, it is certain that no one is ever entitled to promise freedom from upper respiratory infections as a result of tonsillectomy. Too often this promise is given only to have it act as a boomerang. There are biased individuals who believe that tonsils should never come out, just as there are those who need no pretext to order their removal. Somewhere between these two extremes lies the truth, and to him who intelligently weighs the evidence but little difficulty is encountered.

Clinical evidence does not support the view that tonsils are defensive outposts of extreme importance. As a matter of fact more often than not they are so weak that they become an offensive instead of a defensive mechanism.

In certain quarters, there is a feeling that when 60 out of 100 children, reach the age of 11 without tonsils, that too great a zeal has been exerted in having them removed. This feeling may be, in part, a backwash from those tonsil drives which

certain communities have launched from time to time and which usually bring but little credit to the medical profession. It may be partly from disappointment in not having achieved the high hopes held out for relief or prevention of such infections as rheumatic fever. No matter what the cause of the feeling is, most pediatricists who have practiced long enough to have followed a number of children from infancy to adolescence, have a greater feeling of security about their patients whose tonsils are out and not for one moment does the fact that 60 per cent of 11-year boys and girls are without tonsils cause them any uneasiness.

There are problems connected with tonsils that at times cause the pediatricist much distress. He knows for instance that there are tonsils being removed every day either with or without sufficient evidence by incompetent operators. It is no secret that many tonsils are being taken out by men whose operative skill is beyond question, but who perhaps, are not quite as zealous as they might be in their pre-operative preparation or whose postoperative care never extends to the point where later observations are made as to just what is happening in the healing area. No carping criticism is intended by these statements. The pediatricist, perhaps more frequently than anyone else, must give ear to the bitter complaints of disappointed mothers who have been promised the sun, the moon and the stars only to be told that the tonsils have grown back again, that recurrences of rheumatism are not unknown and that the child who did not eat before tonsillectomy may have the same distaste for food long after the operation was performed.

In the millennium there can be no question that children will be operated on only after proper indications are apparent, that preparation will consist of more than a cursory bleeding time and urinalysis, that operative procedures shall make hemorrhages and lung abscesses a thing of the past and never again shall any tonsillar stump remain anything but a stump.

The relationship of the diet to the occurrence of upper respiratory infections has assumed an importance, that, while it is out of all proportion, must nevertheless engage our attention. For many years pediatricists have been giving generous doses of vitamins A and C and D to infants and children. It is a fetish with them. After these years it is now possible to

state that rickets and scurvy can be prevented, but not upper respiratory infections. This, in the face of the far flung claims for cough drop manufacturers and the sacrifice of 10,000,000 guinea pigs. It would be unfair not to admit the possibility of more satisfactory results if doses were increased beyond those ordinarily given in private practice, but overdosage is not a sin of the competent physician.

There is another angle of the diet and nutritional state which is seldom considered, in trying to evaluate vitamin therapy. To do so, however, seems to substantiate the statements just made. It is not too fanciful to assume that bright, active boys and girls enjoying, so far as can be judged, optimum health, have had the requisite amounts of vitamins. Yet these children fall victims to colds just as readily as those not in as good a state of nutrition provided they are not actually suffering from rickets or some other disease.

Another truth must be apparent from this last fact. What holds true for the vitamins probably holds true for the other food constituents whether they be organic or inorganic. Today men and women are being exhorted to dance over on the alkaline side because there lies protection from colds. Ever since the days our ancestors jumped around in trees we have been on the alkaline side, and it has not been of much help. Especially has this been true once the race began to live in places where temperatures drop 40° over night, where the wind gets to hurricane proportions in a few hours and an intermittent sleet and snow sends us home, chilled to the marrow, ready and anxious to get a hot drink. It is much to be feared that the so-called accessory food substances play but a subsidiary part in the prevention of the upper respiratory infections.

These restrictions, however, are not meant to imply that the pediatricist undervalues the necessity of securing and maintaining good nutrition. He asks rather a revaluation of all factors concerned with the prevention of the upper respiratory infections, the putting of the first things first. He asks that children be freed from all physical defects, especially those of the nasopharynx; he asks that children get sufficient rest and that they be kept out of crowded places. He asks that children be protected against colds and dampness and last, but by no means least, he begs adults with infections to stay away from children, especially those under 5 years of age.

The treatment of the early stages of an upper respiratory tract infection is not one of a choice of nasal drops, of packs, of blowing out nasal passages or of bringing them to an office crowded with afflicted children or adults, for any special procedure. It is a question of getting children to bed in a well ventilated room at about 70° F. with normal humidity and forcing fluids, particularly the fruit juices.

If ever a subject were loaded with dynamite, it is the one connected with the question of when should an ear drum be incised. This question is faced as frequently by the pediatricist as by anyone else. His has been a unique opportunity because of the advantages accruing from seeing the child from the start, with the usual simple coryza.

The introduction of the electric otoscope was not an unmixed blessing, although the advantages far outweigh the disadvantages. In the early years of its use, chiefly because of magnification, deepening the color and the fact that pediatricists were seeing ear drums in a fashion that they were not accustomed to, practically every congested ear drum was incised nor did the very simple bleb formations escape a similar fate. This was a mistake and is a mistake. As time has gone on a more conservative attitude has developed and today most pediatricists demand at least a semblance of bulging before incising. Due to a variety of causes, varying all the way from maternal pleas, "Do not cut because the child might become deaf," to paternal commands that you can't cut unless you guarantee no mastoid will develop, pediatricists have watched ear drums bulge and recede to normal just as they have watched spontaneous ruptures follow an uneventful course. This, naturally has tended to confirm the wisdom of conservatism but, as always, there is the ever present danger of the pendulum swinging too far.

The limitations of time forbid the discussion of many other factors concerned with the problem of infections of the upper respiratory tract. For this opportunity to express the importance of them, to ask an added interest in the old problem of the tonsils, to discuss the influence of diet and to bring up for discussion the ever debatable question of otitis media, the speaker wishes to thank the section.

50 East 72nd Street.

SYMPOSIUM ON UPPER RESPIRATORY TRACT AND SOME OF THEIR COMPLICATIONS.

III. — ENDOSCOPY IN THE TREATMENT OF DISEASES OF THE UPPER RESPIRATORY TRACT.*

DR. JOHN D. KERNAN, New York.

The title of this paper should really have been the "Treatment of complications of diseases of the upper air passages by means of endoscopy," as, of course, endoscopy would have no place in the treating of the diseases themselves.

The subject is so broad, that there appears to be great danger of my remarks dissolving into clouds of gas, unless I limit them to a description of specific cases which I myself have encountered.

In a certain number of conditions of the upper air passages, one of the most pressing complications will be acute infection of the larynx with abscess formation and threatening strangulation from the swelling. So, first, then, I will deal with a number of cases in which acute abscess of the larynx has complicated conditions of the upper air passages.

The first case to which I wish to call your attention is that in which the trouble started with a nose bleed. Ordinarily, one would say that an endoscopy could have no place in the treatment of a nose bleed, but this happened to be a very severe one in the case of a man, age 60 years, a nephritic, with fairly high blood pressure. It began spontaneously during a game of bridge. It was so severe as to call for a post-nasal plug and tight packing of the nose. These measures were very effectively carried out, and the hemorrhage stopped. In a couple of days it was possible to remove the packing, and all seemed well. On the fifth day, however, the man began to

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complain of sore throat, which made swallowing very difficult; and finally dyspnea began. It was at this time that I saw him. Inspection with the laryngeal mirror showed such a condition as you see in this slide. You will observe the great swelling of the epiglottis, the ventricular bands, and the right arytenoid. He was taken to the operating room, and under local anesthesia a direct laryngoscope was passed, picking up, not the epiglottis itself, but the base of the tongue. Incisions were made in the most prominent swellings of this man's larynx with the release of scanty pus. In 24 hours he was completely relieved. Cultures show that it was streptococcus hemolyticus. This man, after his laryngeal suppuration, went on and had a very severe case of erysipelas, from which he ultimately recovered.

I should like to digress here for a moment to mention the precautions to be taken in this procedure. A lighted bronchoscope should always be at hand for passing through the larynx, should the choking become dangerous; and a tracheotomy set should also be prepared. Then, if the manipulations of the endoscopy bring on severe dyspnea and cyanosis, the bronchoscope can be passed and a tracheotomy be done at leisure. Anyone who has done a tracheotomy in a patient dying of strangulation will appreciate the advantage of having all of the dyspnea relieved.

Laryngeal abscess frequently complicates infections of the nose and throat. I have seen several cases which began as acute tonsillitis. To illustrate this point, I should like to call your attention to the case of a young girl, age 13 years, whose trouble began with an ordinary sore throat. She seemed to be doing very well, when on the fifth day there was a sudden rise of temperature, and 12 hours later there were signs of laryngeal obstruction. Under local anesthesia her larynx was exposed, showing big swellings of all of the structures of the right side of the larynx. Incisions were made, as you see illustrated in the lantern slide. By the next morning her dyspnea was completely relieved. She went on, however, to have a complication which sometimes arises in these cases of laryngeal abscess, that is, an abscess in the pyriform fossa. The slides which I present now illustrate the situation. This condition continued to develop in severity until it reached the situation shown in this next slide. You will note that there is almost no swelling in the larynx itself, but great swelling

in the region of pyriform fossa and lateral pharyngeal wall. She also developed induration and tenderness in the upper part of the neck on the right side. The situation of the pus and the course it took are illustrated in this next slide. Here was a case in which external incision was necessary.

The danger of the invasion by pus of the deep fascial planes of the neck need hardly be emphasized. I have seen one patient in whom such an invasion took place who died of mediastinitis. This slide will demonstrate the route taken by the pus. The abscess forms in the pyriform fossa, within the thyroid cartilage. This section of the larynx better illustrates the condition. The pus breaks through the cricothyroid membrane and is then under the great vessels with an open pathway to the chest.

In the case of the young girl just reported, operation immediately after the invasion of the neck prevented the pus from reaching the chest. You will see at once the advantage of preventing any invasion of the neck. An operation has been devised for draining the abscess when it first forms in the pyriform fossa. This consists in making a transverse incision over the thyroid cartilage of the affected side, stripping back the soft parts and perichondrium, and then trephining the cartilage. When a sufficiently large opening has been made, an incision is made through the inner perichondrium. The pus will be found between the perichondrium and mucous membrane. If it is asked why such an abscess can not be drained by an incision from above, experience teaches that such an incision fails. I have tried a number of times. The explanation, of course, is that an incision from above is at the very top of the abscess and the pus will not drain up hill. The slide I now show illustrates the suggested operation.

I wish now to say a few words about the mishaps following tonsillectomy which are amenable to endoscopy treatment. First, I shall mention lung abscess. I believe, even in the best of clinics, there will be one lung abscess to every 5,000 tonsillectomies. They start in the first week or two with rising temperature, cough, and later foul sputum. Lung abscesses following tonsil operations are unusually amenable to bronchoscopic drainage. Whether this is because they are due to aspiration rather than embolus, and are near the larger bronchi, I can not say. Only once have I removed an aspirated bit of tonsil from a bronchus. At any rate, such lung abscesses

often get well promptly. Here is a case. This woman developed a lung abscess within a week following tonsillectomy. She had one bronchoscopy and soon was well. Here is a second picture shortly after the bronchoscopy. So, I strongly urge prompt bronchoscopy for lung abscess following tonsillectomy.

Other mishaps occur during tonsillectomy. During such an operation on a woman, age 40 years, a piece of a Hurd tonsil separator broke off and disappeared. Before the patient left the hospital X-rays were made which were pronounced negative. Nevertheless, the woman became quite ill with a cough, attacks of fever, shortness of breath. More X-rays were taken and again were negative. Only after a year was the foreign body found on the film. This was because it had moved from the line of the spine. I urge on you, when confronted with a like situation, not to trust negative X-rays; do a bronchoscopy if there is any question. Also have the films made from several directions. Contrast *these two*, the picture from the front scarcely shows the foreign body, a tooth, whereas, this one from the side shows it plainly.

In the next case I acted on my own advice and did a bronchoscopy though the film was negative. This boy lost a tooth during a tonsillectomy. A film was at once taken, and it showed the tooth. That evening, just before he was bronchoscoped, another film was taken and no one could be sure of the tooth. Nevertheless, he was bronchoscoped, and no tooth was found. He had coughed it up during the day.

Here is another curious sequence of tonsillectomy calling for tube work. This boy, age 9 or 10 years, about two weeks after operation developed fever, stiffness of the neck, dysphagia, and finally dyspnea. Examination showed marked prominence of the larynx and trachea. The lateral films of the neck showed a great thickening of the tissue between esophagus and vertebra. At the time this picture was taken, he also had induration and tenderness in the right side of his neck. He was esophagoscoped, and a general thickening and induration of the posterior wall encountered. It was thought he had an abscess, and the question of drainage arose. Although we freely incise the postpharyngeal wall, incising the esophagus seemed another matter. External drainage was proposed.

While this was being considered, the boy suddenly began to get well. It was thought he might have ruptured the abscess, but examination showed no site of the break.

Here is another retroesophageal abscess. This lady swallowed a piece of glass. She was fluoroscoped promptly, and, as there was no check in the swallowing of a bismuth capsule, it was thought the glass had, for better or worse, gone on down. However, she developed fever and much pain on swallowing. Then, five days after the accident, she had a tube passed. No trace of glass and no wound of esophageal wall could be found. The X-rays told quite a different story, showing a very considerable retroesophageal swelling, which increased for several days and at this stage seemed to show a cavity and fluid level. Again the question arose of infra-esophageal drainage. Much can be said for or against such a procedure. To me it seems dangerous unless a fistula for a lead can be found or a very marked prominence. However, as in the last case, a spontaneous recovery occurred in this one.

Tonsillectomy is not the only operation in our field which may be followed by pulmonary suppuration. In the case of this boy it followed a turbinectomy under local anesthesia. He was very sick for three months in spite of the best of medical care. When he was finally bronchoscoped, his case was quite desperate, but he finally made a good recovery.

The other form of pulmonary suppuration occurring in connection with our specialty is bronchiectasis. It appears as though bronchiectasis almost always had its origin in a sinus suppuration. And one might say that there is little to be done for a bronchiectasis until the nasal suppuration is under control. Even then it is rare to see one which can be cured by bronchoscopy. The odor and secretion diminished, yes, but cured very rarely. Here is one, which owing to special circumstances could be more than a little helped. In this case there was a very tight stricture in the lower right bronchus. This stricture was dilated through the bronchoscope and drainage thus promoted, considerable improvement resulting.

I ought to say a word in regard to hay fever and asthma, and polyps and asthma, and sinusitis and asthma. I personally have never been able to relieve asthmatics by bronchoscopy. Perhaps some of you have, and I hope you will tell your

experiences. There is, of course, a great field for investigation here.

One more case. Five days after an appendectomy, a child, age 5 years, developed a massive collapse of the right lung. Here is the X-ray picture. On bronchoscopy the right main bronchus was found full of thick pus. Aspiration of this, and of plugs coughed from large branches, resulted in a prompt clearance.

120 East 75th Street.

SEMON LECTURE.

This annual Lecture will be delivered on Thursday, Oct. 31, at 5 P. M., at the Royal Society of Medicine, 1 Wimpole street. The Lecturer, Sir St. Clair Thomson, has chosen as his subject: "The Defenses of the Upper Respiratory Tract."

COUGH CONSIDERED FROM AN OTO-LARYNGOLOGICAL VIEWPOINT.*

DR. LOUIS H. CLERF, Philadelphia.

The occurrence of cough as a symptom of pathological conditions involving the upper air or food passages has been commonly observed. Its investigation therefore becomes a problem for the otolaryngologist as well as the internist. The terms *chronic cough*, *winter cough*, *ear cough*, *sinus or nasal cough*, *bronchial cough* and others give remarkably little information concerning the etiology. Their employment as diagnostic terms should not be encouraged. Cough should be regarded as a symptom irrespective of its consideration by the otolaryngologist or the internist.

Cough commonly occurs as a symptom of disease; however, it may be induced by stimulation of certain sensory fibres in the absence of disease. The latter is often referred to as "reflex" cough. Cough is commonly a purely reflex response. The reflex is usually always reinforced by volitional effort. For our purposes it will suffice to recall that cough is dependent upon a local irritant which originates nerve impulses that are transmitted by afferent nerves. The reflex arc is completed through motor or efferent fibres innervating the respiratory and laryngeal muscles. If the stimulus is in the tracheobronchial tree the impulses are carried through the vagus nerve to the respiratory centres in the medulla which excite and co-ordinate the spinal centres. There results increased activity of the inspiratory centre, stimulation of the adductor centre of the larynx and increased excitation of the expiratory centre. This is manifested by a deep, often quick, inspiration, momentary closure of the larynx and a sudden expiratory effort. The larynx remains closed during the beginning of forced expiration. When the required intrapulmonic pressure is

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obtained the vocal cords quickly separate and air is forced out with the production of the characteristic cough sounds.

A very important physiological fact, namely, the establishment of a condition of tolerance, must be borne in mind in a study of cough. A condition of tolerance is promptly established that lessens or entirely obliterates temporarily the reflex excitability of the cough excitation areas. As examples may be cited the tolerance established for the O'Dwyer intubation tube when introduced into the larynx and the absence of cough often observed following the initial paroxysm of coughing, choking and gagging in laryngeal and tracheobronchial foreign bodies. This has been corroborated by all bronchoscopists. Whether this results from exhaustion of the end-organs in the mucous membrane is a question.

Since cough is a symptom one must ascertain its cause in order to treat it intelligently. The older clinicians attached much importance to the character of the cough and this gave rise to innumerable descriptive terms. All of us recognize the dry and the productive cough. This classification is not dependent upon the patient's ability or lack of ability to expectorate after coughing. Expectoration is an accomplishment that comes from practice. Young children and many women invariably swallow secretions that are coughed up into the pharynx. Paroxysmal cough, as the name implies, occurs in fits or paroxysms. It may be dry or productive. The brassy, metallic, croupy or laryngeal cough is commonly the result of irritation of the nerve endings in the larynx or trachea. It may be associated with changes in the size of the airway, as in subglottic swelling, stricture of the trachea or a bronchus and aneurysmal compression of the trachea.

What are the causes of cough? It would seem unnecessary to consider the common respiratory diseases of which cough is a prominent symptom. There usually is no difficulty in ascertaining the causes of cough in patients with pulmonary tuberculosis, abscess of the lung or other pulmonary diseases that can be demonstrated by routine Roentgen study and physical examination of the chest. I shall therefore consider the less obvious and probably less common causes which are of interest to the otolaryngologist. We are all familiar with the so-called "ear cough" or reflex "aural cough," which often

can be provoked at will by irritating the external auditory canal. This results from stimulation of Arnold's nerve, the auricular branch of the pneumogastric (certain authors mention the auriculotemporal nerve). In discussing cough as a symptom of impacted cerumen, Dench emphasized the importance of examination of the ear in the investigation of every case where complaint is made of cough alone. Cough may also be observed in eczema, local inflammation and foreign bodies in the external auditory canal. These may be considered as extrarespiratory causes.

Cough is often noted in diseases of the nose and nasal accessory sinuses. It may be a symptom of acute coryza, simple chronic and hyperplastic rhinitis, spurs and deflection of the nasal septum, enlargement of the middle turbinates, polyps, contact between the turbinates and the nasal septum and in vasomotor disturbances. Certain "cough spots" in the nose have been described. Among the more common are the "tubercle" of the septum and the posterior and anterior ends of the inferior turbinate, although in certain patients the middle turbinate is equally sensitive. Another common site of origin lies in the "trigger area" of swallowing at the base of the tongue, supplied by the superior laryngeal nerves.

The mechanism of cough production in nasal disease is not clear. It is not a constant symptom. Patients with extensive nasal disease may have no cough whatever. In other cases remarkably slight nasal disturbances will be promptly followed by cough. In many individuals introduction of a cotton-tipped applicator will provoke paroxysms of cough. In these one can definitely demonstrate that there must be cough excitation areas since cocaineization promptly gives relief in many cases. The cough is usually dry and may or may not be paroxysmal. In children it is more often paroxysmal and is worse at night. The relationship between sinus disease and lower respiratory tract infection is generally recognized. It is not uncommon to observe patients with cough who have been considered as cases of tuberculosis, although positive findings to support the diagnosis have never been secured. In these cough is persistent and is not controlled by the commonly employed sedatives. Careful examination in certain of these reveals unsuspected sinus disease, treatment of which is followed by relief of cough. In cases of laryngotracheobronchitis or bron-

chiectasis with sinus disease the cause of the cough is more apparent.

Cough may occur in the presence of adenoids, acute or chronic pharyngitis, paralysis of the soft palate and elongation of the uvula. One must always remember that interference with nasal respiration necessitating mouth breathing is a not uncommon cause of symptoms referable to the tracheobronchial tree. Enlargement of the lingual tonsil due to hypertrophic changes or the occurrence of neoplasm or inflammation in this locality are common causes of cough, probably through irritation of the nerve endings of the superior laryngeal nerve.

The effects of certain habits unquestionably exert an influence on cough by reason of circulatory and mucosal changes. The cough of the chronic alcoholic results from local congestive changes in the pharynx, larynx and trachea, together with certain systemic effects that undoubtedly are responsible for the chronic inflammatory changes of the mucosa. Smoking sets up chronic congestive changes of the mucous membranes. There is an increased activity of the secretory cells with the production of thick, tenacious mucus. This is difficult to dislodge and gives rise not only to cough but frequent clearing of the throat. Persons using alcohol or tobacco excessively are often troubled with morning cough, particularly after taking food or fluid. Exposure to dust and to fumes exerts an unfavorable influence on the mucous membranes of the respiratory tract, resulting in chronic congestion and production of cough.

Posture at times exerts a very definite influence on cough. This may be explained on physical grounds in cases of pulmonary abscess, tuberculosis or bronchiectasis. Patients with bronchial neoplasms, aneurysm or other mediastinal and pulmonary lesions often must lie on one side or be propped up by pillows because of the occurrence of cough if another position is assumed. Cough occurring in a patient free from pulmonary disease is not always so easily explained. Among the more common causes are sinus disease, elongated uvula, enlarged tonsils, hypertrophied lingual tonsil, adenoids, pharyngeal diverticulum and esophageal stenosis involving the upper esophagus. Certain individuals start to cough on getting into bed. The nose, throat and laryngopharynx should

be carefully searched for a possible cause. Cough may be caused by congestion of the mucous membrane of the nose, throat or larynx, due to chilling of the skin by the cold sheets.

Cough of functional origin does occur; one must be exceedingly careful, however, in excluding organic disease as a cause. Nervous cough is observed in children, particularly boys at puberty, and is often barking in character. Cases of functional aphonia frequently complain of cough. By mirror laryngoscopy one observes the usual laryngeal picture on phonation; that is, inability to approximate the cords normally, but on coughing the vocal cords are seen to approximate properly. The characteristic cough sounds are normal, in striking contrast to the sounds produced in complete abductor paralysis of the larynx.

Cough is very frequently initiated by laryngeal or paralaryngeal disease. It may result from foreign bodies, inflammation or neoplasms of the larynx. It is often observed in abductor paralysis and usually results from overflow of secretions. Paralysis of the superior laryngeal nerve, sometimes injured during thyroidectomy, must be considered. The relationship of cough and the taking of food should be inquired into. Overflow of food or secretion may occur in sensory or motor paralysis of the larynx, esophageal stenosis, pharyngeal diverticulum and esophageal fistula communicating with the trachea or left bronchus. It is important to distinguish between *cause* and *effect* when considering inflammatory conditions of the larynx as the cause of cough.

How should one then proceed in an investigation of this symptom, cough? A careful study of the onset and character of the cough, the question of expectoration and appearances of the sputum, the time of occurrence and associated symptoms are important. Information secured from the history will aid in directing subsequent investigations.

A study of the chest should be made to rule out pulmonary, mediastinal and cardiac diseases. Obviously the more common causes of cough should be excluded first. One should then proceed with examination of the ears, nose, mouth, throat, pharynx, larynx and neck. If no cause can be found in a routine study of this character bronchoscopy is indicated. In

certain cases a functional study and esophagoscopy may be necessary.

Treatment should be directed towards removing the cause; one must not ignore the patient while this is being accomplished. The patient may be contented with the effects of a cough sedative; the physician, however, should be interested in cause as well as effect. The use of narcotics to control cough is always a problem. Cough is necessary to rid the tracheo-bronchial tree of excessive secretions, as in pulmonary abscess or bronchiectasis; in these, narcotics should be used sparingly, if at all. Hypnotics that do not obtund the cough reflex are preferable. In a dry cough, sedatives may be employed. All of the potential sources of irritation to the mucous membranes of the air passages should be interdicted. This includes smoking. Local applications to irritated and inflamed mucous membranes will give temporary relief. Intralaryngeal instillations of medicated oils are very useful. All forms of local medication should be bland and soothing. The important considerations in treatment are removal of the cause and giving relief to the patient.

1530 Locust Street.

THE AMERICAN BOARD OF OTOLARYNGOLOGY.

An examination was held in New York, June 8, 1935. One hundred and nine candidates were examined, of which number 85 were certified and 24 were conditioned or failed.

The Board will hold an examination in Cincinnati, Ohio, Sept. 14, 1935, prior to the meeting of the American Academy of Ophthalmology and Otolaryngology. Prospective applicants 1500 Medical Arts Building, Omaha, Neb., for application for certificate should address the Secretary, Dr. W. P. Wherry, blanks.

INJECTION OF IODIZED OIL INTO THE BRONCHIAL TREE. PASSIVE METHOD THROUGH NOSE.*

DR. HENRY M. GOODYEAR, Cincinnati.

It is agreed generally today that there are frequent uses for the injection of iodized oil into the bronchi, both for the Roentgenograms and for the treatment of bronchiectasis.

If frequent injections are to be made, it is necessary that the method be simple, requiring no unusual technical skill, and is not objectionable to the patient.

Forestier has recently demonstrated the method of pouring iodized oil through the nose after cocainizing the larynx and bronchial tree as follows: the tongue is drawn forward (see Fig. 1), the head extended and 5 cc. of a 1 per cent solution is injected through the nose. Five minutes later this injection is repeated and again in five minutes, making three injections in all. Considerable of the solution is expectorated; however, it is well to test the patient against cocain prior to the injections.

The oil should be injected within three minutes after the last 5 cc. of cocain, since the swallowing reflex is only temporarily abolished. The larynx is fixed and the upper esophageal orifice remains contracted. The extreme extension of the tongue pulls the epiglottis and soft tissue forward, bringing the open glottis in position to receive the oil as it flows down over the soft palate.

The injections, except for treatments, are made before the fluoroscope. The patient is tilted slightly to the right, injecting 10 cc. of the oil into the right bronchus, and then to the left, injecting 10 cc. into the left bronchus (see Fig. 2).

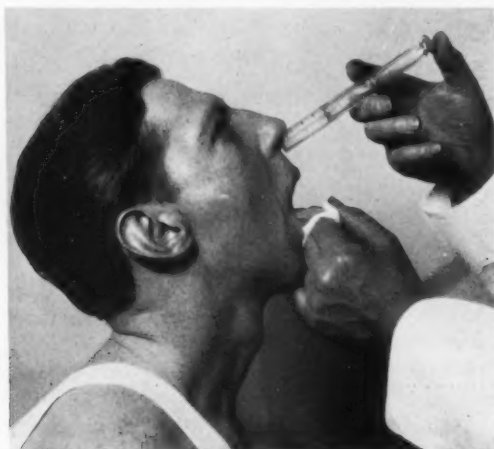
To inject the right upper lobe the shoulder and right arm hangs over the table with the patient in the reclining posture and the hips slightly elevated. While in this position the head

*From the Ear, Nose and Throat Service, Christ Hospital, Cincinnati.

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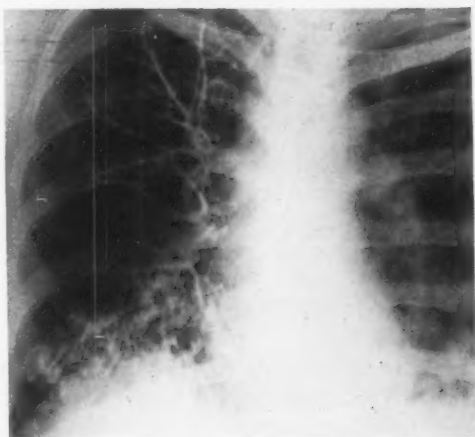
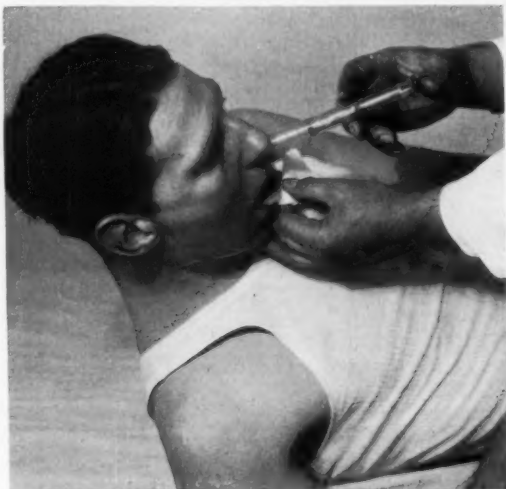
is raised and the injection repeated as in Fig. 3 flowing into the right upper lobe (see Fig. 4).

I have found that a No. 16 French catheter can be inserted through the nose and into the trachea without special guid-



ance after cocainization as for the oil. I have also passed a Levine stomach tube after the same manner, turning the

patient to the side on which the bronchus is to be entered. This might be a very useful method in sucking out the bronchi in



a postoperative atelectasis, where it would be difficult or inadvisable to subject the patient to bronchoscopy. By the use

of an opaque tube the procedure can be observed under the fluoroscope.

Immediately after the injection of opaque oil in the lungs the Roentgenograms in a normal chest show that the alveoli have been filled to the extent that the outline of the bronchi is concealed. Thus the picture represents a tree in full leaf in summer with its branches concealed. The reverse of this picture is true in a case of bronchiectasis, where the distinct outline of the bronchi resembles a tree in winter bare of leaves.

Any branches which retain the opaque oil 15 minutes after injection is functionally impaired.

Repeated injections of iodized oil in cases of bronchiectasis at intervals of five to seven days, then at longer intervals, improves the condition of the patient, who often gains weight, coughs less, and is generally more comfortable.

Patients having a bronchitis extending over months or recurrent attacks of bronchitis should have an opaque oil injection and Roentgenographs made, thus many beginning cases of bronchiectasis may be discovered.

In cases of hemoptysis where the usual radiograph does not demonstrate pathology an oil injection should be made. Thus an unsuspected bronchiectasis is often revealed as the cause of the bleeding.

CONTRAINDICATIONS.

1. Acute or subacute inflammations with or without temperature as the progress might be extended.
 2. Weak myocardium where increased dyspnea and pulmonary edema might prove embarrassing.
 3. Extensive emphysema and pulmonary fibrosis if the heart is bad.
 4. Never in early lung abscesses, as the progress may be extended.
 5. Hyperthyroidism.
 6. Chronic asthmatics may react badly.
- 556 Doctor's Building.

THE INJECTION OF THE SPHENOPALATINE GANGLION.*

DR. SIMON L. RUSKIN, New York.

Blocking of the sphenopalatine ganglion for a variety of conditions, ranging from hemicrania of the so-called "lower half headache" to vasomotor states, such as hyperesthetic rhinitis and asthma, was practiced by Sluder over 25 years ago. In performing this procedure, Sluder injected 0.5 cc. (7 drops) of 5 per cent phenol in 95 per cent alcohol intranasally, lateral and behind the posterior tip of the middle turbinate.

In 1924 the author first introduced the technique of blocking the sphenopalatine ganglion by injecting through the roof of the mouth at the posterior palatine foramen into the palatamaxillary canal, which, serving as a grooved director, leads the needle directly to the sphenopalatine ganglion. This technique has the following advantages:

1. It is independent of nasal abnormalities, *e. g.*, septal obstructions, polypi, turbinate malformations, lateral wall variations.
2. It is less likely to be followed by hemorrhage, because the injection is in the depth. The nasal area is difficult to approach for the control of bleeding.
3. It is more accurate, because the canal guides the needle.
4. It is much simpler and can be performed by anyone familiar with the regional anatomy.
5. It is safer, because it cannot enter a danger zone if the correct depth of needle is used. The nasal route permits the passing of a needle through the posterior corner of the ethmoid into the cranial cavity, with accompanying dangers. The risk of postinjection sloughing is inherent in the use of alcohol as a medium for injection rather than the type of approach.

Indications: The indications for the injection of the sphenopalatine ganglion are as follows:

Since the sphenopalatine ganglion, as the chief nerve centre of the nose, serves as a crossroads for three different nerve

*Exhibit for the Inter-State Postgraduate Medical Assembly of North America, International Meeting, Philadelphia, 1934.

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tracts, there are accordingly three different groups of symptoms possible, as well as mixtures of all three. These are:

1. The trigeminal group, which supplies by way of the maxillary nerve the two or three sphenopalatine nerves which pass through the sphenopalatine ganglion.

2. The facial group, which supplies through the greater superficial petrosal part of the Vidian nerve which enters the sphenopalatine ganglion.

3. The autonomic group, which supplies sympathetic fibers through the filaments that encircle the internal carotid as the deep petrosal part of the Vidian nerve which enters the sphenopalatine ganglion. Craniosacral autonomic traveling with the greater superficial petrosal.

Inflammatory or other processes affecting the sphenopalatine ganglion may thus give rise to the following conditions:

On the part of the *trigeminal* system: 1. Maxillary neuralgia, or major trigeminal neuralgia. The frequency of purely maxillary neuralgia has given rise to the misnomer of "lower half headache."

2. Pains referred to the nose, sinuses and orbit.

On the part of the *facial* system: 1. Pains referred back to the geniculate ganglion at the middle ear by way of the greater superficial petrosal.

2. Reflex muscle spasms affecting groups innervated by the facial nerve; *e. g.*, frowning muscle, orbicularis oculi, corrugator supercilii and frontalis, causing so-called eye headaches.

3. Ear noises, stuffiness, impairment of hearing from tubal and ossicular disturbance.

On the part of *autonomic* system: 1. Vasomotor disturbances, from overaction of the craniosacral autonomic, hyperesthetic or vasomotor rhinitis, nasal response to allergy, asthma; from overaction of the vasoconstrictors, ozena or milder forms of "dry nose."

2. Muscle tonus disturbances remote from nasal areas. The autonomic nervous system as a diffusely acting mechanism is

apparently easily affected in a general way through medication or shock of one of its various ganglia. In spastic muscle states, such as torticollis, lumbago, writer's cramp, anginal pains, the relaxation of the spasm is rapidly effected by topical blocking of the sphenopalatine ganglion with saturated solution of cocaine, butyn, silver nitrate, etc. Of these cocaine is the most effective.

3. Tearing, from cranial autonomic passing to zygomatico-temporal nerve from sphenopalatine ganglion.

4. Dysmenorrhea, from craniosacral autonomic influence.

CONTRAINDICATIONS.

1. Asthma; although theoretically particularly indicated and useful in some cases, the author has observed in two asthmatic cases sudden arterial spasm, affecting the internal maxillary artery in one case and the external carotid in the other, with subsequent sloughs in the former of the palate soft tissue and in the latter case of the cheek and ala of the nose.

2. Myocardial weakness.

CAUTIONS.

1. Do not inject both sides simultaneously, to avoid sudden interference with circulation to palate. Inject one side and second side a week or more later. Do not perform a third injection within a month. Too frequent injection may cause slough of soft tissues.

2. Do not inject rapidly. Use only 0.5 cc. (7 drops) at each injection. This will avoid diffusion upwards toward orbit, which may cause abducent paresis.

3. Do not insert needle to full length. Always leave 0.5 cm. above ring exposed; this brings depth of injection to 3 cms.

4. Do not angle needle laterally. Keep vertical to palate, angle slightly towards nose.

DIRECTIONS FOR INJECTION (SITTING POSITION).

1. Anesthetize sphenopalatine ganglion in the nose with one-half drop of saturated cocaine solution on cotton tipped appli-

cator (Ruskin model) placed under posterior tip of middle turbinate for one to two minutes.

2. Swab side of roof of mouth to be injected with 4 per cent solution of cocaine.

3. Paint area to be injected with S.T. 37.

4. Under strictly sterile precautions palpate hamulus of pterygoid process. Posterior palatine foramen lies about 1 to $1\frac{1}{2}$ cms. anteriorly.

5. Watch for pale spot or dimple alongside of second molar tooth close to where soft palate color changes to hard palate color, about 0.5 cm. medial to alveolar process.

6. Use tongue depressor to secure more room for syringe.

7. If needle strikes hard palate, do not force, but probe gently without removing needle tip.

8. If slight bleeding occurs, change needle to keep it clear.

9. Put patient to bed in sitting position.

REACTIONS.

1. Slight to moderate swelling of cheek.
2. Numbness of maxillary nerve distribution in some cases.
3. Rarely abducent paresis, twice in over 1,000 cases.
4. Superficial sloughs, four times in same series of cases.
5. Ecchymosis of cheek and lower lid, twice in same series of cases.

There were no hemorrhages, no fatalities, no orbital complications.

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351 West 86th Street.

THE USE OF ULTRA SHORT WAVES IN SINUS THERAPY.

CONRAD K. GALE, New York.

The use of short and ultra short waves in medicine is relatively recent. Being a new modality, there is very much unknown both as to its physiological and physical action in the body. The object of this paper is to discuss its use and effects upon sinus infections.

In December, 1927, it was found that workmen while testing radio equipment had a rise of temperature. The temperature rise was found resulting from the radio frequency field surrounding the sending apparatus. This led chiefly to the experimenting on rises of body temperature, using wave lengths between 25 and 50 m. The so-called "fever machines" were used specifically for their heating effect and the physiological rise in temperature was the weapon used to combat the disorder being treated.

In America, some work had been done on the short wave, relatively little on the ultra short. It must not be forgotten that diathermy is also a high frequency wave with its main field of usefulness in the approximate 500 m. range. The spectacular difference between the short waves and diathermy consists in that short waves can be used upon acute and sub-acute infections which is contraindicated to diathermy because of the danger of exacerbations and recrudescences.

To determine the frequency of a wave, its length in meters is divided into 300,000,000 m., which is equivalent of 186,000 miles per second, the velocity of light. Thus a 10 m. wave has a frequency of 30,000,000 cyc. per second and a 1 m. wave would have a frequency of 300,000,000 cyc. per second. The shorter the wave length becomes, the more rapid is its frequency of oscillation; and when the wave length has shrunk to that of .02 cm. we speak of it as infrared.

It must be apparent that radio waves are the longest waves in the ether spectrum and that a 10 m. wave, although called

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an ultra short, is an ultra short wave only in comparison with a 1,000 m. or longer wave length. Since the 10 m. ultra short wave belongs in the long wave length part of the ether spectrum, we find that when the wave length really becomes relatively short, as for example, that wave length seen by the eye as red, its frequency of oscillations have already mounted to 400 million million cyc. per second. The name ultra short is therefore seen to be a misnomer, since in reality an ultra short wave is one of the longest in the ether spectrum. By a short wave, is arbitrarily meant one between 10-20 m. long. Ultra short waves are below 10 m.

Are ultra short waves harmful?

From the position of short waves in the long end of the ether spectrum the answer theoretically would be no. (X-rays occupy the short end of the ether spectrum, and are cited as an example of the caution with which this end of the spectrum is to be treated. Ultra short waves, being longer than the recognized innocuous infrared rays, would therefore be even safer to handle.) From the practical experience of workers in this field it is definitely recognized that ultra short waves from 4 m. up are harmless, even on prolonged exposures. Personally after continuous exposure for $1\frac{1}{2}$ years to ultra high frequency fields, I have noticed no ill effects either to myself or patients. As far as I know, wave lengths from 3 to 4 m. are also harmless, but the amount of work done in that range is relatively small and so must be a partly moot point at present.

The power of the ultra short wave apparatus is also of prime importance. An U.S.W. of 10 m. and 200 watts power will have a much greater effect upon the body than the same wave length at 100 watts. For this reason it is important in treating a patient to know not only what wave length is being used but also the power back of the wave. Since the patient is being treated with the power coming out of the apparatus, we are concerned therefore only with the power output. An apparatus may have a 500 watt input rating; that is the power in the machine mounts to 500 watts. However, that rating is useless from the medical viewpoint, since the patient is to be treated with the power which comes out of the machine. Depending on the wave length generated, as much as 75 per cent of the power generated in the machine may be lost. There-

fore, in judging the intensity of an U.S.W. treatment we must know how many watts the apparatus delivers at a definite wave length into the patient circuit.

Since we are treating the head we must place our condenser plates from the apparatus equidistant from the cheeks, or if an anterior posterior treatment is given, equidistant from occiput and front of face. Withdrawing the head from the field, let us see what electrically happens when the wave is generated between these two plates. If a 10 m. wave is being generated it is obvious at once that it is physically impossible for a 10 m. wave to exist between two plates 1 m. apart. What does exist is a high frequency field having the same number of oscillations per second that its respective wave would have. In the above illustration while a 10 m. wave does not exist, the high frequency field is oscillating at the rate of 30,000,000 cyc. per second, which is the oscillatory rate of a 10 m. wave. These oscillations take the form of parallel (open to question) electrokinetic lines of force traversing the field between the two plates. When the head is placed in this field, it distorts this pure electrical field absorbing within itself the power of the field. The head now acts as a conductor of these electrokinetic lines of force and we find that within the head are induced electric currents of the same oscillatory frequency as those existing in the field. We now come to the problem affecting the rhinologist. What is the effect upon the sinuses?

To affect the sinuses we must have penetration of our ultra short wave. That ultra short waves penetrate into the tissue depths is now an accepted fact. Let me state here, however, that it is not the ultra short wave that penetrates but the induced electric currents. These induced currents oscillating at millions of cycles per second have certain peculiarities which determine the depth of penetration; namely, a selective heating action on tissues determined by their conductivity and dielectric constants. The following experiments explain these points.

Experiment 1: Three glass dishes are so placed within each other that their sides do not touch. The inside two rest on cork so that there is no contact anywhere. This gives us three spaces, an outside, middle, and inside. The outside space is filled with tap water, the middle space with 0.1 per cent potassium permanganate, and the inside space with 3 per cent salt

solution. An 8 m. wave is used with 300 watt output. Temperature at start of all three solutions, 78° F. The plates are put 1½ in. from the sides of the outer dish. At the end of 10 minutes:

Temperature of outer space, 79° F.; temperature of middle space, 81° F.; temperature of inner space, 93° F.

Here we find a selective action of the induced frequencies for the innermost space, although it is surrounded by two layers of fluid, which the induced frequencies must have traversed. The reason for this selectivity is the higher conductivity, and dielectric constant of the 10 per cent salt solution.

Experiment 2: The outer space is now filled with 3 per cent salt solution, the middle with 0.1 per cent salt solution, and the inner space with tap water. Eight m. wave at 300 watts used. Temperature of all three spaces, 78° F. After 10 minutes' exposure:

Temperature of outer space, 93° F.; temperature of middle space, 84° F.; temperature of inner space, 80° F.

Here we find the outer space, because of its higher conductivity and dielectric constant, absorbing the induced frequencies and acting as a shield to the deep penetration of the wave. It is this shielding effect that is of paramount importance in the body. Because of it, we cannot reach any deep organ with this modality if it is surrounded by a tissue layer which can act as a shield.

Experiment 3: A patient has a permanent opening into the left antrum. Two condenser plates are put 1½ in. distant from cheek; 300 watts at 8 m. Mouth temperature at start, 98.5° F. Temperature of air in sinus, 98.5° F. The sinus opening was plugged with cotton. Patient was exposed for one-half hour, at the end of which time mouth temperature was 100° F. The thermometer inserted into sinus showed temperature 99° F. Because of mechanical difficulty the thermometer could not be inserted far enough into the sinus to touch a wall and so determine that temperature. Ringers' solution was now put into the sinus and the patient exposed with the head held to the left. Unfortunately as the temperature was being taken after 15 minutes' exposure, the patient moved his head, the solution ran out of the sinus and its temperature was not obtained. Lack of co-operation made further tests impossible.

However, the above experiment shows that the interior of a sinus does not heat up on exposure to even 300 watts. The cause for this lies in the fact that it is impossible for ultra short waves to penetrate through a sinus. The induced currents flow around the *walls* but do not traverse the sinus. The reason for this lies in the fact that the air content of a sinus has a lower conductivity and dielectric constant than the walls. If the sinus were filled with pus or exudate, then according to the physics of ultra short waves we would expect to find penetration of the induced currents through the exudate, with a subsequent rise in temperature.

In presenting the following clinical cases it will be noted that all are chronic.

Case 1: R. L., adult male, has had a pus discharge from the right nostril for the past eight weeks. Less discharge from the left nostril. Pain over the right cheek and frontal region has been severe for four weeks. X-rays show definite involvement of the right ethmoid and both frontals. Ethmoidal polyps on both sides seen. Much pus seen. Took routine treatments without result, previously. Ten m. at 300 watts given with plates 2 in. from the sides of the cheeks. After first treatment pain much relieved. After four daily treatments, pain gone, no discharge. Patient has remained well until present writing. Duration of treatment, 45 minutes.

Case 2: L. M., age 5 years. Both nostrils stuffed for past year. Purulent discharge. X-rays show bilateral chronic ethmoiditis. Much purulent discharge seen in both middle meati. Has been treated for past six months without any results. Using above routine, the condition cleared up entirely within two weeks and a total of eight exposures. Patient has remained well for the past seven months.

Case 3: A. P., adult male. Has had frontal headaches for past year. Much purulent discharge. X-rays taken six months ago show marked clouding of both frontal sinuses. There is exquisite tenderness on slight pressure over both frontal regions. There is a marked purulent discharge in the nose. Was treated for several months previously with no result. Operation advised. A 10 m. wave with 200 watt output was used and the plates adjusted in the anteroposterior position. Immediately after the first treatment (20 minutes) the pain left. Subsequent treatments were extended to 45 minutes.

Pain did not return after the fifth treatment. In all, 10 treatments were given over a period of three weeks. To date, three months after the last treatment, the patient is symptom free and no purulent discharge is seen in the nose.

The above cases illustrate the types treated. I believe it is as yet too early to go into the causes for the results obtained, but from the experience gleaned from treating 50 chronic sinus conditions I believe that the ultra short wave to be the best means of combating sinus infections at present. Of course, our results were not 100 per cent perfect, but then each case treated acted as its own control since other treatment without result had been previously given in all cases.

We believe for good results an ultra short wave apparatus should have a minimum of at least 200 watts output. The position of the plates should be equidistant from the sides of the cheeks, or in the anteroposterior position. Until more is known about the modality it is impossible to control the penetration of the wave by manipulating the electrodes. It is best to keep them equidistant from the area being treated.

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40 West 86th Street.

ATROPHIC RHINITIS-OZENA WITH SPECIAL REFERENCE AS TO TREATMENT.

DR. PHILIP S. STOUT, Philadelphia.

My experience with atrophic rhinitis-ozena dates back more than a quarter of a century and from that time to this I have pondered a great deal over this strange illness, which causes a constant worry and chagrin to the unfortunate sufferers. This foul carrion-like odor is ever present, following them like some evil spirit. Naturally, I have gathered much data on this subject during these years but it is necessary that this thesis be kept within reasonable limits, so I will consider only one phase of this subject, namely, some of the various treatments I have tried and the results obtained therefrom. A number of these cases were treated in the Nose and Throat Clinic of the Jefferson Hospital under Dr. Chevalier Jackson and later under Dr. Fielding O. Lewis, during the late war at Camp McClellan, and prior to that time under Dr. R. H. Skillern and Dr. C. P. Grayson.

In cleansing the nostrils and posterior nares various saline solutions were used.

These were used in one way or another since the first case I ever treated. 1. Plain water douche — this will do but is not as good as 2. Salt water douche, half normal solution, or 3. Salt and iodine douche, half normal salt solution with tr. iodine, gtt. x to quart of saline solution. 4. Diluted sea water itself is most excellent for cleansing the nostrils.

In passing, it might be mentioned that Gen. Gorgas used this in atrophic (ozena) cases during the building of the Panama Canal and this has since then been spoken of as the Gorgas treatment. When sea water is used it does help and occasionally relieves the patient completely. Incidentally, the writer got this information some years ago from a Dr. Young (a descendant of Brigham Young, of Utah), who was in Panama when the treatment was used. Gen. Gorgas noticed

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that when a sufferer with ozena took frequent ocean baths, getting the salt water up his nose, he was much better, so he simply had some sea water — if too strong, diluted with water — used in the clinics and from these treatments had some very pleasing and lasting results in his ozena patients.

I have used these solutions and found them very satisfactory. Of course, sea water is not easily obtained, so I use half normal salt solution, 1 or 2 qts.; tr. iodine, 10 drops to 1 qt. of water.

Douche the nostrils, head hanging well forward, once or better twice daily. Incidentally, not one case of otitis media has developed during these years of douching nor has any case of sinus disease developed by douching, that I know of. Other cleansers may be used:

Solution permanganate of potash — wine color — as a douche, this controls the odor and is helpful.

Hydrogen peroxide can be applied by the physician to help remove the crusts, but do not advocate its use by the patient at home.

Dobell's solution, half strength, as a spray is soothing and cleansing; oil of mustard, 1 drop to 1 or 2 oz. of a bland oil makes a good stimulating application.

Cleansing should be your first thought in the treatment of ozena and it will go a long way to help the patient bear the horror of this disease. Incidentally, suicide is not so uncommon among these sufferers, so make them as comfortable as you can; assure them that patients have been greatly relieved and at times cured. This takes a great weight off their minds and they are grateful to you and they are more liable to continue to follow your instructions if they have some hope of ultimate relief.

Vaccine Treatment. The writer first used this in 1917 on some clinic patients. This vaccine was made by Parke, Davis Co., but never marketed. Later, during the war, 1919, I was at Camp McClellan, in the Nose and Throat Department, and it so happened that the ozena patients were referred to me. Those in the service, who had ozena or other foul odors such as bromidrosis, were not permitted to go abroad unless the condition was made less objectionable to others in their

company. I wrote to Parke, Davis Co. and explained the circumstances and they very kindly sent me about a pint of ozena vaccine, which I used on quite a number of ozena cases that occurred in the 35,000 officers and men of the Twenty-ninth Division stationed there at that time; and, as far as I was able to learn, no one failed to go abroad because of foul odor from his nose. I wish to take this occasion to thank the above firm for their co-operation in this matter and say that a vaccine made from all of the germs found in the noses of ozena patients does, at least temporarily, relieve the foul odor, but that later it recurs.

Internal Secretions: Unquestionably the internal secretions have something to do with this disease, coming as it usually does at puberty, lasting indefinitely for 15 or 20 years or more. At times, as if by magic, it suddenly ceases, or at least the foul odor diminishes; and while the nostrils are just as wide, there is no odor, although small crusts may form. The writer has used thyroid and mixed glandular treatments with doubtful results. In passing, it might be stated that in my experience, young girls of the Latin races are more likely to suffer with this disease, although during the war we saw quite a number of men; but, in looking back over the patients, they were those men who were frequently of the slightly feminine type; that is, they more resembled their mothers than their fathers. I treated one family of a mother and two sons, all suffering with ozena. At the age of 30 years all three recovered completely. I, therefore, took some blood from these patients on several occasions and, with the aid Dr. Kolmer at the laboratory of the University of Pennsylvania Graduate Hospital, the serum was separated from the cells and this made sterile and injected into six patients, into the muscles of the back, beginning with 5 cc. or 10 cc. doses and increasing the dosage — noting the reactions, if any — for four or five doses. This was done in 1930. These half dozen patients showed immediate good results, but the bad cases that we had under treatment for a long time finally came back with a recrudescence of the odor and crusts. The milder cases seemed to be free from the trouble; at least they did not return as they were asked to do should foul odor return.

Operations: Needless for me to say that when the ozena is unilateral, and I had a number of these, should there be a

deflected septum, making one nostril much wider than normal, a submucous resection is very beneficial and almost always curative.

Other operative procedures have not been of much avail; in fact, I have resolutely refused to do some of the nasal narrowing operations as recommended because in all I have seen, following operations, the patient was no better and the nostril horribly deformed; and to do this on a young girl is beyond the comprehension of the writer.

Tonsils had better be left alone — the throat will not become quite so dry if the tonsils are not removed.

If the condition is recent and unilateral, I have on several occasions found it to be due to sinusitis, usually maxillary or ethmoidal. Clearing up this condition of the sinuses relieves the ozena at once, but I have not encountered this condition bilaterally. In ozena the sinuses are frequently quite clear and free from infection, as shown by transillumination and X-ray.

Although the writer is connected as Rhinolaryngologist in the Allergic Clinic at Jefferson Hospital under Dr. J. Alexander Clark, where he has seen between 3,000 and 4,000 allergic cases, he does not recall a hay fever patient with ozena, although he has a record of a number of bronchial asthma cases with ozena.

Unilateral ozena may be due to syphilis. Wassermann and the sloughing off of a turbinate or a septal perforation confirm the diagnosis. Antiluetic treatment is indicated. Incidentally, mercury and iodide of potash are still to be thought of if the arsenicals fail to relieve.

Diet: This is a big factor in treatment. No doubt the starches have much to do in increasing the severity of this condition. By cutting down on starches, especially spaghetti and macaroni, and sugars, much relief will be found.

Local Applications: After you have treated the patient with mild solutions: argyrol, 10 per cent, or iodine, iodide of potash, glycerin, 10 gr. to fl. oz.; or tr. benzoin and boroglycerol, equal parts, you can gradually increase the strength until you can use a combination of the three tinctures: tr. benzoin comp., tr. iodine, tr. guiac, equal parts applied to nasal mucous membrane and back of throat. This burns pretty badly, and possibly you should use a local anesthetic for the first two or

three times, but, after that, the writer has not found it necessary. Patients come back for this treatment over and over again.

Emulsion of scarlet red has been recommended, but the writer did not find it any better than the above treatment. Lightly plugging each nostril with cotton plugs, with or without a mild antiseptic, has certainly helped certain cases.

Use of Filtrates: Prepared in the laboratory of Jefferson Hospital by Dr. Crawford. Filtrates are made by using the crusts and also from swabbed material from the mucous membrane of the nostrils. This is grown in broth for about a week and then it is filtered through a Berkfeldt filter. This is injected under the skin or applied directly to the mucous membrane of the nose. I have done a number of these but found that there were, again, immediate good results and then a recrudescence some time later.

Accoline Injections: I have used this preparation as prescribed on several patients with perhaps slight improvement, but the patients developed other unpleasant symptoms. I do not think I will continue its use on others.

Silver Nitrate — Silver Oxide Colloidal: Applying silver nitrate in varying strengths has helped in some cases. Recently, silver oxide colloidal, 5 per cent solution, has been tried, but cannot say that much has been gained by its use.

Vitamins: It is just possible that the lack of some vitamin may account for this condition since, when vitamin A is completely removed from the diet of say 100 chicks, a rather large number die and show degenerative changes in the brain. This is not often found in chickens with the usual diet containing vitamin A. Reasoning along the same line, it occurred to me that possibly there was an imbalance between the vitamins and the internal secretions at puberty, thereby causing an atrophy and degeneration of the nasal mucous membrane, throat, and on down into the stomach. I had a number of my patients examined and there was also some atrophy in the rectum and lower sigmoid. I am now trying some vitamin stimulation. However, it is too early to report.

Mecholyl: Formerly called mecholin (acetylbetamethylcholine chloride-Merck).

The writer has used this 0.5 of 1 per cent solution, sprayed over the mucous membrane every other day. Again there seems to be some change (slight) that seems to be beneficial.

Ionizing of this material into the mucous membrane has been tried.

I hope that in time this or some other similar substance may help in overcoming this vessel constriction, thereby relieving this condition.

Short Radio Waves: Department of Physiotherapy, Jefferson Hospital, in co-operation with Dr. Schmidt. Short radio waves are now being tried for so many different chronic conditions. I have tried these waves on a few ozena patients, but it is too soon to make any deductions.

One patient had 48 treatments without any marked improvement.

In conclusion, make your patients comfortable first, then assure them that much can be done to help persons who have such a condition and that from time to time patients are entirely relieved. This will be correct and will give your patients confidence, for the permanent relief of a bad case of ozena is particularly difficult and you must not leave any stone unturned or any method unused to relieve these unfortunate sufferers. I hope that before long this enigma of ozena is solved.

269 South 19th Street.

VOCAL NODULES AND CROSSED ARYTENOIDS.

WILLIAM A. C. ZERFFI, New York.

While a considerable amount of information regarding the formation of vocal nodules is available, the writer believes that there are certain causative factors which have by no means been investigated with sufficient thoroughness. The consensus points to incorrect voice production as the chief cause, but the expression "incorrect voice production" is too indefinite to offer a satisfactory solution. Since nodules occur chiefly among those who make use of their voices as a means of livelihood, the matter ceases to be of mere academic interest, it is a vitally important question and one which should be fully answered.

Singer's nodules are described by Herbert Tilley as follows: "A 'singer's node' is usually seen as a small white and sometimes translucent swelling upon the free edge of the vocal cord, and situated at the junction of the anterior with the middle third of the cord; as a rule both cords are affected." That the primary cause is irritation has been established beyond reasonable doubt, additional proof being supplied by the fact that unless the nodules have become well organized and are of long standing, rest will invariably effect a cure. It is also of interest to note that they are far more prevalent among women than men.

During the singing of low tones, the vocal cords are not closely approximated, and it is only when the attempt is made to sing the higher tones that the cords are brought more closely together. There is therefore no doubt that it is the singing of high tones which is responsible for the irritation which results in the nodular formation. When a normal effort is made to raise the pitch of the voice, the cords are approximated and tensed by the combined action of the arytenoid muscle, the lateral cricoarytenoids, and the thyroarytenoids internally, and by the cricothyroid, the palatopharyngeus and stylopharyngeus, externally. The action of the arytenoid,

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the lateral cricoarytenoids, and the thyroarytenoids is that of bringing the posterior edges of the cords closely together and thus shortening the vibrating length. This approximation helps to raise the pitch of the tone, and high tones are thus sung with the anterior two-thirds or even with the anterior half of the cords. Since nodules occur at the junction of the anterior third of the cords, high tones sung as described could not possibly produce an irritation of the anterior third. Nor are there any laryngeal muscles which act in such a manner as to provide sufficient pressure at this point to result in the formation of nodules. It is therefore obvious that other muscles than those of the larynx proper are concerned in this action. In the opinion of the writer, the muscles which bring about this pressure are those which are concerned in deglutition. These muscles, notably those of the tongue, the action of which can be detected by means of finger palpation, assist those of the larynx in bringing about complete closure of the larynx when deglutition is about to be performed. Forced production of the voice is brought about by similar action as that employed in deglutition. A partial contraction of the muscles is used to force the vocal cords into approximation and only by the generation of considerable energy can the breath be driven through the glottis. It would appear that during this action the anterior third of the cords remains closed, the tones being produced by the vibration of the posterior two thirds. This would account for the formation of the necessary nodal point at the junction of the anterior third. Careful finger palpation during deglutition and forced voice production will unfailingly reveal the similarity of action. Additional proof of the validity of these contentions is supplied by the fact that nodules are more prevalent in females than in males. According to V. E. Negus (*The Mechanism of the Larynx*), upon laryngoscopical examination: "the vocal cords of man are seen to shut with a snap on making an effort with the arms in an adducted position—in the case of women, closure of the glottis is the exception." This is borne out by observations made by the writer who has seen a continuous forcing of the female voice result in a chronic separation of the vocal process, whereas in the case of the male voice, continuous forcing often leads to a crossing of the vocal process, known as crossed arytenoids. While the superior strength of the arytenoid muscle is undoubtedly the determining factor in this

latter disability, aid is no doubt furnished by the muscles of deglutition referred to above. This type of forced singing in the female voice does by no means always lead to the formation of nodules, the writer having observed many cases where the muscular action was greatly disturbed, but the cord tissue unaffected. In fact it was noticed that the cords of those who were able to produce tones of unusually fine quality, implying pliability and sensitiveness of cord tissue, were far more likely to be susceptible to the formation of vocal nodules.

Many years of observation and experiment have convinced the writer that the attempt to solve the problems of both the occurrence of vocal nodules and crossed arytenoids without taking into consideration the action of muscles other than those which directly affect the action of the vocal cords is doomed to failure. Furthermore, finger palpation in effecting a diagnosis is absolutely essential, for it offers a definite means of determining the action of extrinsic muscles, which if not inhibited in action, inevitably bring on serious vocal troubles.

246 West 71st Street.

PRESENT STATUS OF MASTOIDECTOMY.

DR. WESLEY C. BOWERS, New York.

In reading the history of mastoidectomy, it is astonishing to find that keenness of observation and profound knowledge existed hundreds, even thousands, of years ago. Many of the arguments of those times in regards to mastoids and mastoidectomy sound strangely familiar and some of the present day observations and "discoveries" are of remarkable antiquity. Even Hippocrates knew that a recurrent infection in an ear was more serious than a primary infection and that one in a sclerosed bone was more apt to be fatal. Two thousand four hundred years ago he wrote: "Acute pain in the ear, with continued strong fever, is to be dreaded, for there is danger that the man may become delirious and die. * * Younger persons die of this disease on the seventh day or still earlier, but old persons much later * * in the later periods of life relapses of the disease coming on generally prove fatal."¹ Celsus, nearly 2,000 years ago, observed: "If the discoloration (in a mastoid) extends right through the bone, it must be excised and all disease must be extirpated * * cauterization is useless. Excision is imperative."² The first authentically successful mastoidectomy was performed about 1740 by a French surgeon, Jean Louis Petit. In this connection he states: "We ought not to wait until the pus has destroyed the bone, for the patient is always in danger, not only because of the pus which does not escape, but because of the accidents that may supervene and render the disease infinitely complicated and fatal."³ The anatomy of the mastoid bone was well known before 1863, but Schwartze, in that year, was the first, as far as we know, to establish drainage through the mastoid to the antrum.

While it would thus seem that we have been merely repeating ourselves for hundreds of years, nevertheless I should like to emphasize a few points bearing on the present status of

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mastoidectomy. I shall not touch on the variations of the surface anatomy, the position of the sinus in relation to other parts, the grouping of cells in different types of mastoids, nor shall I burden you with the well known technique of mastoidectomy.

The more or less successful results in the past depended on varying degrees of thoroughness, but the percentage of successful operations was much lower than at present, chiefly because incomplete removal of cells frequently led to complications and secondary operations. Today those familiar with the anatomy of the mastoid bone and its surroundings and variations are usually able to eliminate every approachable cell and mastoidectomy, by its brilliant results, may be considered a real triumph in surgery. Postoperative reaction is slight in proportion to the amount of work required; the dressings are not painful; the wound heals solidly in from three to six weeks, leaving a scarcely perceptible scar and, in most cases, very little depression; the tympanic membrane and hearing return to normal. The great improvement in postoperative treatment is another favorable factor.

The weak point in simple mastoidectomy is the impossibility of reaching directly the cells beyond the labyrinth and in the middle ear. Undoubtedly the deep cells in the petrous and in the surrounding parts of the middle ear may be involved as well as the mastoid itself. In a simple mastoidectomy we do not remove the cells around the middle ear, although they are numerous in the neighborhood of the Eustachian tube, on the anterior and inferior walls of the middle ear, and internal and anterior to the attic. Fortunately, infection at these points clears up in most cases when proper drainage is established through the antrum. A certain amount of drainage to the cells beyond will be provided by opening the cells deep in the mastoid bone and establishing thorough drainage of the attic, but the comparative rarity of petrous bone abscesses and chronic purulent otitis media following mastoidectomy can only be explained by the lack of cellular development at the above points, the slight involvement there, or the good natural drainage through communicating cells.

Many of us can recall cases which have been admitted to the hospital with meningeal symptoms on which we performed a mastoidectomy. While cleaning out the cells around the

semicirculars or along the dural plate of the posterior fossa we have encountered sinuses leading into abscesses from which considerable pus was evacuated. A probe passed into these sinuses seemed to pass to dangerous depths, the sinuses were enlarged as much as possible, and some of the cases recovered. Again, in cases with VIth nerve paralysis where mastoidectomy was followed by a most profuse persistent discharge from the external auditory canal, the wound has been reopened to insure that no cells have been overlooked; nothing has been found; the discharge from the middle ear has continued for a time, then ceased and the patient has recovered. In still other cases the mastoid wound has healed completely, yet the patient has died of fulminating meningitis several weeks or months later.⁴ In the light of our present knowledge we realize that all of the above cases had abscesses in the petrous bone. In this connection I should like to emphasize the following points: 1. Although some operators prefer to disturb the antrum as little as possible, I consider it essential to clean it out thoroughly, and especially to eliminate the cells above and internal to it, as drainage from the petrous bone and middle ear is obtained from this area. 2. All cells around the semicircular canals and those extending along the dural plate of the posterior fossa should be carefully followed. Cells along the former frequently lead to abscesses within the petrous bone; cells along the latter to abscesses on the posterior surface of the petrous bone. 3. The cells which often lie between the descending limb of the sinus and the facial nerve should be carefully searched for and eliminated since these may, in turn, lead to a large collection of cells internal to the sinus, between it and the jugular bulb. This is a point favorable to abscess formation, because of the restricted outlet between sinus and facial canal, on the under-surface of the petrous pyramid. If we were more careful to eliminate cells at these three places we should be successful in establishing drainage of the cellular involvements beyond the confines of the mastoid.

After completion of the operation *per se*, a careful inspection is made of the sinus and the dural plate. If the latter seems too thin or if there are any suspicious signs it should be removed, since frequently a diseased sinus or a collection of infected secretion is found under an apparently healthy plate. When possible, exposure of sinus or dura should be

delayed until the end of the operation, and they must be guarded against trauma caused by handling or by resting drains on them.

The after-care of the mastoid wound varies considerably. Some operators still leave the wound open, packed with gauze; others prefer to sew it up almost completely, leaving a small rubber drain in the lower angle. Between these two extremes there are many variations. It should be taken into consideration that with gauze packing there is very little drainage after the gauze is saturated; indeed, there may be danger of its acting as a plug. The wound is sometimes cleaned out with drugs, suction or irrigation, but such interference, with its risk of secondary infection, should not be necessary in a well cleaned out mastoid unless there are complications. It is always unsafe to close the wound too completely or to try to hasten healing unduly. Too quick healing means either that the operation was unnecessary or that Nature has already done most of the work with the exception of one or two places where there has been abscess formation; the latter is usually true in cases of at least five weeks' duration. Then, too, premature healing interferes with drainage of the deeper cells.

In complicated cases it is essential that the wound be left wide open; in uncomplicated cases we prefer a method which has been used for 15 years in hundreds of cases with very satisfactory results, both from the surgical standpoint and that of the patient's comfort. It consists in suturing the subcutaneous tissues and skin to within $1\frac{1}{2}$ in. (in large mastoids) from the lower angle of the wound. Dead spaces should be obliterated. Rather heavy rubber dam drain is then placed in the wound in such a manner that it extends up to the antrum, spreads out in the wound, and is brought out through the opening in the lower angle, folded over on itself two or three times. Rubber dam, used in this way, will not injure exposed dura or sinus as gauze or rubber tubing are apt to do; it affords excellent drainage and can be handled without pain to the patient. This drain is loosened daily, shortened very slightly and removed at the end of a week, leaving a good sized opening through which the cavity may be inspected. A new and smaller drain is now inserted. This procedure is repeated until it is considered safe to allow the wound to close. It is important that this drainage be promoted and pro-

longed over a period sufficient to avoid complications due to the impossibility, already noted, of removing all cells by a simple mastoidectomy. There may be involvement of the remaining cells of the middle ear and in the petrous bone, but there is hope that this will subside because of the improved drainage that has been established. The sooner the wound heals the greater is the danger of these deep cells not recovering, especially in cases where the bone is markedly pneumatic. The healing of a mastoid wound within a few days or a week reflects no credit on the operator.

At the time of operation the auditory canal is packed very firmly in order to hold the fibrocartilaginous canal against the bony meatus. This packing is left undisturbed for a number of days, the time depending upon the amount of drainage through the meatus. This packing and the preservation of the bony posterior canal wall prevent the falling forward of the fibrocartilaginous posterior canal wall and the resultant narrowing of the external auditory meatus, which has so often proved troublesome in after years.

One important factor in the successful outcome of mastoidectomy must not be overlooked; namely, the opportune time for the operation. A decision on this point requires the keenest judgment and, when rightly exercised, lessens the possibility of needless operation, reduces the number of complications and paves the way for smooth convalescence. From the standpoint of the general reaction of the patient and the healing process of the wound itself, the least favorable time to operate is during the period of invasion, *i.e.*, a week or more from the onset of the original ear infection; the most favorable time is after Nature has had a chance to throw out her barriers and wall off the infection. If it were possible always to wait until this latter period has been reached the end-results of mastoidectomy would be greatly improved, but unfortunately there are occasions when early interference seems imperative. Invariably in such cases there is more reaction, convalescence is less smooth, the wound does not heal so well, and complications are more frequent. While it is true that in some cases complications may be due to the severity of the infection or to poor general resistance, there is no doubt that in others they are caused by the trauma of operation at a time when the channels of extensions are still open. I believe

that if the number of complications occurring spontaneously during the first few days of a mastoid involvement were compared with the number of those following mastoidectomy performed in the first few days of an infection of the mastoid, the greater number would be found to occur in the operated cases. On the other hand, where there is a history of repeated attacks, the more recent the previous attack the earlier is operation justified.

No hard and fast rules can be devised either for the necessity of an operation or for the opportune time to choose. A mastoiditis showing every sign of fulminating involvement will very often clear up without operation; whereas operation may be urgently indicated in cases without physical signs and symptoms and in which the results of laboratory tests and X-ray examinations are negative. Each case presents an individual problem. We must recognize the conditions which add to the danger of a case: the appearance of the patient and his reaction to the infection; the influence of age; the peculiarities of anatomical formation, such as the presence or absence of sclerosis, the type of bone and pain; the position of the sinus; the relation of the point of greatest destruction to important structures; the influence of previous attacks; the characteristic reaction of certain organisms; the significance of the blood examination; the presence of renal disease or diabetes. From our study of the whole picture we must visualize the pathological conditions present at the time of our examination and we should not be deceived by any of our findings, including the X-ray. If there is any reasonable chance of localization we should wait for it. The final decision, however, depends on clinical experience, judgment and, sometimes, intuition.

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17 East 61st Street.

HEMOLYTIC STREPTOCOCCUS MENINGITIS OF OTITIC ORIGIN. OPERATION, RECOVERY.*

DR. ARTHUR J. CRACOVANER, New York.

Cases of streptococcus meningitis which have recovered are still rare enough to warrant the report of this additional case. It is encountered with such infrequency that J. A. Kolmer¹ said that the mortality in streptococcus meningitis is nearly 100 per cent. Applebaum² in 1932, reported 46 cases of streptococcus meningitis that recovered, including three of his own. He included in this report the cases collected by Rosenberg and Nottley³ in 1931. Of these 46 cases, 29 were of otitic origin. Felsen and Osofsky⁴ collected 11 more cases and reported one of their own and mentioned still another in a report in 1934. One case each has been reported by Kleinfeld⁵, Newman⁶, Ebert⁷ and Rochett⁸.

In a recent symposium on meningitis, Neal, Applebaum and Jackson⁹, studied 623 cases of meningitis excluding meningococcic and tuberculous types. Of the total, 16 patients recovered. Of 121 streptococcus infections due to otitis media, mastoiditis and sinusitis, nine recovered.

CASE REPORT.

W. P., male, white, age 4½ years. Admitted to Dr. J. Leopold's Service at the Lenox Hill Hospital on May 29, 1934, with a chief complaint of vomiting, drowsiness and an earache of 12 hours' duration.

Present Illness: Child had had an earache and discharging ear on the right side for two weeks. Discharge had stopped two days before admission. On the morning before admittance to the hospital, child began to complain of pain in the ear,

*From the Service of Dr. John D. Kernan, Lenox Hill Hospital, New York City.

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began to vomit everything he took by mouth, and appeared very drowsy. He was seen in the afternoon by his physician who sent him to the hospital immediately.

Previous Illnesses: Ever since child was nine months old, he had been subject to upper respiratory infections which were accompanied by otitis media. In December, 1933, he had an attack of otitis media followed by scarlet fever two weeks later and a mastoidectomy had to be performed on the left side. Three weeks later, the mastoid had to be revised. The child was well until three weeks before admission, when he developed a bilateral otitis media and both ear drums were incised. The left ear was well again within two weeks, but the right ear continued to drain until two days before admittance to hospital. The rest of the history was negative.

Examination: Well developed and well nourished child appeared very drowsy and restless. Temperature of 105° F. The skull and scalp were negative. Eyes reacted to light and accommodation. Eye grounds were normal.

Ears: Right ear drum was a little full and red, but not really bulging and some of the landmarks could be seen. No drooping of the canal wall. No mastoid tenderness. Left ear drum normal. *Nose:* Negative. *Throat:* Acutely inflamed. *Neck:* Slight rigidity. No Kernig or Brudzinski. No cervical adenitis. *Chest:* Clear. *Heart:* Negative. *Abdomen:* Negative. *Genitalia:* Negative. *Male.* *Extremities:* No abnormalities. *Back:* Negative. *Reflexes:* Knee jerks hyperactive and equal. *Abdominals:* Hyperactive and equal. *Babinski:* Negative.

Diagnosis: Acute otitis media, right, and a questionable meningitis.

The next day, Dr. Julian Rogatz examined the child and reported that the throat was acutely inflamed. Child was drowsy and irritable. Temperature was 104.4° F. Rigidity of neck, tache cerebrale. Bilateral Kernig. Right O.M.P.A. He advised spinal tap.

Spinal Tap: Twenty-five cc. cloudy fluid not under tension removed; 20 cc. antimeningococcus serum given. Count showed 1,350 white blood cells, polys, 70 per cent and 30 per cent lymphocytes. Smear revealed an occasional gram positive dip-

lococcus. Blood count was 22,400 leukocytes with 91 per cent polys.

Nose and throat consultation was called. The ear was slightly full. There was no discharge. The short process could be seen. There was no mastoid tenderness. The neurological signs were noted as above. Temperature was 104.8° F. Mastoidectomy was decided upon and done at 9 P. M.

Findings at Operation: The cortex was intact. Mastoid cells were broken down. Pus and granulation tissue were found especially in the upper portion of the mastoid. The necrotic process involved the tip and sinodural angle but there was an especially soft area in the zygomatic region near the dura. This seemed to be the only possible place of extension if the extension was a direct one from the mastoid. Dura was seen to be markedly inflamed and bulged.

On May 31, the next day, temperature came down to 101° F. General condition was considerably improved.

Spinal Tap: Thirty-five cc. cloudy fluid was removed under pressure. Spinal fluid cell count was 1,750 with 73 per cent polys.

On the following day, the spinal tap again showed 2,050 white blood cells and 88 per cent polys and a culture subsequently revealed hemolytic streptococcus. Temperature ranged about 102° F.

On June 2, spinal tap showed 1,450 cells, 90 per cent polys and again hemolytic streptococcus culture.

On June 3, he again had a positive culture in his spinal fluid. Subsequently no positive growth was obtained in the spinal fluid though he had been tapped daily until June 13. The cell count of the spinal fluid gradually diminished. With the increase in the cell count and the finding of a positive organism in the spinal fluid, the sugar content decreased and the globulin increased. As the spinal fluid again became normal, the sugar increased and the globulin decreased. The mastoid wound was dressed and gradually healed.

On June 14, a transfusion was given. Signs of meningitis gradually cleared up and his temperature gradually came down. Blood culture was negative on several occasions. Sub-

sequently he developed an acute suppurative osteomyelitis of the lumbar vertebrae at the site of the spinal punctures. This was operated upon and after drainage for some time it finally healed. Culture from this wound was hemolytic streptococcus.

Child has gained weight, his appetite is good and he acts as a perfectly normal child would.

Conclusion: A case of hemolytic streptococcus meningitis of otitic origin is here reported recovered after simple mastoidectomy, daily spinal taps and a transfusion.

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THE RELATION BETWEEN THE SHORT AUDITORY MEMORY SPAN DISABILITY AND DISORDERS OF SPEECH.*

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A few years ago it was considered adequate to diagnose any speech defect either as functional or as organic.

It is now known that functional speech disorders are caused in many very different ways, and that the most effective treatment varies widely with the cause. Hence, it is most important that the diagnostician determine the cause of the case being examined, if possible.

If inarticulate speech is due to deafness, the hearing must, if possible, be improved either by adequate medical care or by mechanical means; otherwise speech must be acquired in special classes through other senses, such as sight and feeling.

If a speech defect is caused by lack of unilateral cerebral dominance, exercises must be prescribed to develop such dominance.

If defective speech is due to marked mental deficiency, the child should be instructed in special classes in a school for the feeble-minded.

If a functional dyslalia is caused by a short auditory memory span, speech training must be restricted to a very limited number of the most useful short words most often heard by the child, no word containing more sound units in any one syllable than the length of the child's auditory memory span.

It is this latter cause of functional disorders of speech which I have never seen stressed in print, that I want to discuss.

Psychologists use the term "auditory memory span" to denote the number of presentations that can be returned after one repetition when given orally. If a person is able to repeat

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without error one series out of three equal series of digits, consonants, or nonsense syllables given orally, the longest such series he is able to repeat thus without error is accepted as his auditory memory span. The average 3-year-old child has for digits an auditory memory span of three; that is, he can usually repeat three digits correctly after another person, but cannot repeat four correctly as often as once out of three different presentations. Although the auditory memory span for the average 4-year-old child is four digits, that of five digits is not reached until the seventh year; that of six digits until the tenth year; that of seven digits until the fourteenth year; and that of eight digits until the eighteenth year, and then only by the superior adult. The auditory memory span for logical sentences is considerably longer than that for digits, consonants, and nonsense syllables, being about seven syllables at the age of 3 years, 12 syllables at the age of 4 years, 16 to 18 syllables at the age of 6 years, 20 to 22 syllables at the age of 10 years, and 28 syllables at the age of 16 years, which is the mental age of the average adult.

Although these norms have been determined for the auditory memory span, it is uncertain just what ability is being tested when this span is measured. In a study entitled "Correlation of Auditory Digit Memory Span with Intelligence," Arthur S. Clark¹ found there was no correlation between a poor auditory memory span and intelligence. Auditory memory span merely measures one of the ways that a child gets his facts, and in no way measures how the child may use the facts after he gets them. The child of normal intelligence with a short auditory memory span is thus far superior to a mentally retarded child who easily gets the facts, but can do little with them after he has them.

The Stanford revision examinations of 40 dyslalia cases and of 46 stammerers seen by the speaker at the Habit Clinics conducted by the Division of Mental Hygiene of the Massachusetts Department of Mental Diseases, showed that 10 per cent of these dyslalia cases had superior auditory memory spans, 45 per cent had normal spans, and 45 per cent had short spans for their chronological ages, whereas 20 per cent of the stammerers had superior auditory memory spans, 59 per cent had normal spans, and 21 per cent had short spans. Eight per cent of the clutterers had superior spans, 38 per

cent had normal spans, 38 per cent had spans retarded by one digit, and 15 per cent had spans retarded by two digits. Thus these stammerers averaged normal auditory memory spans, whereas 45 per cent of the functional dyslalia cases had retarded spans.

Short auditory memory spans were found in 21 per cent of the stutterers, in 28 per cent of the pure sound substitution cases that were not complicated by another speech defect such as stuttering or cluttering, in 53 per cent of the clutterers, and in 60 per cent of the delayed speech cases. Only one clutterer had a superior span. There seemed to be no correlation whatever between the degree of unilateral cerebral dominance and the length of auditory memory span. It is unsafe to draw definite conclusions until at least 1,000 speech defectives have been thus tested, but the above results suggest that many functional speech disorders, especially cluttering and delayed speech, may be due primarily to a retarded auditory memory span, and that this problem is of sufficient practical importance to deserve further quantitative study.

Miss Margaret J. Saunders,² one of the psychologists for the Massachusetts Habit Clinics, finds that children who are handicapped with short auditory memory spans present similar traits and characteristics.

"They are children who talk at a late age, although showing normal progress in other forms of development such as walking and dentition; they have speech peculiarities and defects, often language for some time being unintelligible; they are equally slow in acquiring any facility with language; they confuse directions and commissions; they learn with difficulty rote material when given orally; they are not musical; they have difficulty in learning to read; they struggle with phonics; they are poor spellers and their general progress in school is slow.

"There is great similarity also in the social behavior of children with the short auditory span. Invariably they are unassertive, shy and bashful.

"They are usually immature, placing great dependence on others. If he is the first child in the family or the only child he becomes very dependent upon the mother. With others it may be an older brother or sister who assumes the lead

socially. They are easily upset emotionally, this taking the form usually of much weeping."

Miss Saunders also found that these children possess little musical ability, that they are unable to make fine discriminations between different sounds, and that they are unable to give lists of rhyming words.

The auditory sense is the sense most employed in the life of the young child. It is essential in language development, as it is through this sense that the child receives his language patterns. The congenitally deaf never learn oral speech unless taught by some special method. Granted a normal speech mechanism, the first requisite for learning to talk is the ability to carry and relate sound; this necessitates a normal auditory memory span. When language patterns are given in a larger number of sound units than the child can carry and integrate, they inevitably confuse him, because the addition of an extra sound to a language pattern is great enough to throw the whole pattern into confusion. The child is confused by sounds that are not clearly differentiated, wrong associations are made, and poor habits of spelling and enunciation are formed.

Short memory spans are allied with difficulties in reading and spelling, and cause many young children of normal intelligence to be regarded as mentally retarded or even as feeble-minded unless their intelligence is rated by a nonverbal or performance test, rather than by a Stanford-Binet test. The latter does injustice to children who have short auditory memory spans in the early grades, where rote memory plays a more important part than logical memory and the handling of facts. Selinda McCauley³ tested 1,000 children who were unable to make normal progress in school and found a poor auditory memory span was significant of unsatisfactory progress; she maintains that an auditory memory span of at least five is necessary if one is to maintain a place as a normal member of society.

Language is normally learned by imitation of the word patterns spoken by others. The young child learns by imitation the language he hears spoken in his home; if the family speaks English, he will speak English; if the family speaks French, he will speak French; if they speak Spanish, he will speak Spanish.

Since language is learned by sound imitation, children who have a short auditory memory span are at a great

disadvantage in acquiring it. Although they may learn to understand many of the most common words they hear spoken in the home, they are often unable to reproduce words containing three or more different sounds until they are from 3 to 6 years of age, depending on how much their auditory memory span is retarded. As most words in common use contain three or more sound units, these children are unable to reproduce ordinary words. The task of repeating a word after another person seems so hopeless and impossible to them that they will not even attempt it; they take no interest whatever in learning to speak; they seem entirely satisfied to get what they want through gestures. This speech disorder is known as *alalia prolongata* — delayed speech. It is often useless to try to teach such children a vocabulary until they have acquired an auditory memory span of three sound units.

Because conventional language seems an impossibility to young children who are handicapped with a short auditory memory span, some of these children invent a language of their own which bears no relation whatsoever to any conventional language. Such an individualistic language is known as *idiolalia*.

Children who can pronounce correctly by itself every conventional language sound sometimes mispronounce so many words that their speech becomes unintelligible, a speech defect known as *barylalia* or *cluttering*. A child having an auditory memory span of but three sound units usually finds it impossible to pronounce correctly new words or syllables containing four or more sound units or even certain difficult combinations of three sound units such as "try," "claw," "ask."

Speech defects which are caused by a short auditory memory span should be treated by a special system of speech training which takes this handicap into consideration.

It is necessary first of all to determine the child's auditory memory span, so that he may be given only those words which come within the length of his auditory span.

His parents and teachers must speak to him very slowly and distinctly in a single language, pronouncing very clearly every letter spoken, in order to give the child a distinct language pattern. They must speak to him at first in a very few

words which come within his experience, such as the names of his toys, articles of his food and clothing, parts of his body, and verbs which concern these. They must use these words over and over again, and select only those words which do not contain more sound units than the child can carry. After the child has mastered this very limited vocabulary of the most common and essential words, his parents should gradually call his attention to the names of other common objects which have simple names and seem to interest the child.

In the case of delayed speech and invented language, parents and teachers should encourage speech without trying to force it by asking the child to say a particular word. They should let the child play with younger children who speak distinctly, as younger children are apt to use fewer and simpler words than adults, and a child is more apt to imitate the speech of a child than that of an adult. Try to interest the child in games like authors, which require speech, being careful to choose a game that will especially interest the child. Encourage the child to imitate animal calls and noises which he hears; once he begins to imitate these, he soon will imitate words. Never speak for the child or anticipate his wants; do not try to understand his gestures or his invented language after he is able to make the request by means of conventional speech. Listen attentively and patiently to him whenever he attempts to use conventional speech and try to understand him. Never correct him for mispronouncing a word lest this discourage his attempts at speech; it does not matter how indistinctly he speaks as long as he attempts to use conventional speech. If he still mispronounces certain letters or words after he has been speaking for two or three years, he may need the drill outlined under cluttering or paralalia.

In the case of cluttering, encourage the child to speak very slowly and distinctly, sounding each letter. Help him to build up a word which he mispronounces and which contains more sound units than the length of his auditory memory span from groups of sound units within his auditory span. For example, if he omits some of the sounds in the word "streets," start with the word "eat" which he probably knows as a single unit. Then teach the word "treat" as a unit by separating it into the following three sound units, "t-r-eat;" then "street" through "s-treat;" and finally "streets" through "street-s." It is very necessary to pause between each sound unit and to

shorten this pause gradually until the sound units merge into the desired word pattern. Words of more than one syllable usually have to be taught through syllable units, as they contain more sounds than a short auditory memory span permits the child to integrate.

In the case of paralalia, speech drill becomes necessary if the child does not speak distinctly within two or three years from the time when he first begins really to talk. As children who are handicapped with short auditory memory spans usually have abnormally poor ability to discriminate between sounds which sound or feel somewhat alike, they should practice pairs of words which differ only in the correct and incorrect sounds, such as "path — pass," "fit — pit," "tame — came," "march — marsh." After they have learned to make a given sound correctly, they should read aloud sentences which have this sound printed in bold face type of a size smaller than the other letters of the word to warn them in time to shape it correctly. They should then read ordinary print, and finally talk as they read.

An analytical study made by the speaker, of the first 500 words the average child learns, as reported in "A Reading Vocabulary for the Primary Grades" by Professor Arthur I. Gates, of Teachers College, Columbia University, showed that 6 per cent of the monosyllabic words among the first 50 words acquired contained a single sound unit (such as "I"); that 61 per cent had two sound units (such as "me"); and that 33 per cent consisted of three sound units (such as "cat"). Two per cent of the monosyllabic words among the next 50 words acquired contained a single sound unit, 30 per cent had two sound units, 58 per cent three sound units, and 10 per cent four sound units. But 1 per cent of the monosyllabic words among the next 400 words acquired contained a single sound unit, 14 per cent had two sound units, 60 per cent three sound units, 23 per cent four sound units, and 2 per cent five sound units. Two per cent of the first 50 words contained two syllables as compared with 6 per cent of the next 50 words and 18 per cent of the next 400 words. Eighty-five per cent of these two-syllable words were accented on the first syllable. There were no three-syllable words among the first 300 words, and but 1 per cent of the first 500 words and 2.5 per cent of the next 500 words contained three syl-

lables. There were no four-syllable words among the first 500 words, and there were but four in the next 500.

An additional sound unit was added in the syllable of polysyllabic words at about the same time it was added in monosyllabic words. For example, the fifth sound unit first appeared in the two hundred and twenty-first word, "Christmas," (a two-syllable word), as compared with the two hundred and fifty-second word, "drink."

The syllable unit is evidently more difficult than the separate sound unit, as it seems to lag two units behind this. Five sound units were used before a single three-syllable word was acquired, and an equal number of words containing four sound units and of two-syllable words were listed in the first 200 words acquired by the average child.

Groups of consonants are evidently more difficult than the same number of consonants separated by a vowel. But two consonants were adjacent in any monosyllabic word or syllable containing four sound units; these pairs of consonants were equally common at the beginning or at the end of words or syllables. A vowel came between pairs of consonants in all syllables containing five sound units with the single exception of the word "street."

Three hundred and sixty-nine children in the sixth month of the first grade in the Belmont, Massachusetts, public schools were given a list of 125 words. These children recognized 84 per cent of the words containing a single sound unit, 74 per cent of the words containing two sound units, 57 per cent of the words containing three sound units, and 44 per cent of the words containing four sound units.

SUMMARY.

1. Many cases of functional dyslalia, especially of barylalia or cluttering, seem to be caused by short auditory memory spans.
2. Spasmophemia or stammering does not appear to be caused directly by short auditory memory spans.
3. Children who are handicapped with short auditory memory spans should not be taught words containing more sound units than the length of their auditory memory spans.
4. The first 50 words acquired by children who have short auditory memory spans should be interesting monosyllabic

words containing but one or two sound units, such as the pronouns "I" and "me."

5. The next 50 words acquired may contain three sound units if consonants are separated by a vowel.

6. Polysyllabic words must be learned from syllable units rather than from letter units; syllables should at first be separated by a dash in print and by a pause in speech.

7. Monosyllabic words and syllables containing four sound units with not more than two adjacent consonants are not a stumbling block for the average child who has an auditory memory span of four digits and has acquired a vocabulary of 100 words; neither are two-syllable words.

8. If it is necessary that a child learn a word or syllable containing more sound units than the length of his auditory memory span, this must be built up from shorter units within the child's span, as "eat, t-r-eat, s-treet, s-treet-s."

9. As five sound units, three adjacent consonants in a single syllable, and three-syllable words are not found in any of the first 250 words acquired by the average child, and are seldom encountered in the next 250 words attained, they should not be learned until the child has acquired an auditory memory span of five digits, which is normally reached at the age of 7 years.

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THE USE OF UNDENATURED BACTERIAL ANTIGEN (KRUEGER).

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The evaluation of any new therapeutic measure is a very difficult thing. It has been the endeavor of the author to judge the merits of Krueger's antigen as accurately as is possible. Cases have been observed over a year's time, and sufficient controls were utilized at the time of giving the antigen to accurately check on the therapeutic effect.

Krueger has attempted to find an antigen that would be a good immunizing agent. He found that heat and chemical treatment of bacterial suspensions, as ordinarily employed to kill the bacteria in making a vaccine, produced serious alterations in the protein structure. In endeavoring to find the method which would not alter the antigen, or as is commonly spoken of, denaturate it, he employed a physical disruption of the bacteria in a specially built ball mill. The product is called undenatured bacterial antigen, or simply UBA; and will be so referred to later in this paper.

UBA is in no way connected with the bacteriophage. It is not the living phage that will lyse organisms, and thus clear broth cultures of suspended bacteria. It also will not inhibit growth of bacteria on culture media when they are exposed to its action. Thus we see UBA has no lytic principle, and no bacteriostatic principle at its command.

In making UBA, Krueger attempted to produce a product which was free from all metabolites, and broth proteins; in other words, free from any foreign nonspecific protein. He also attempted to obtain a material which was as nearly free from denaturization as possible. Another desirable result was to have a product that would have sufficient concentration to be active enough to immunize the host against the invading

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organisms. Following these ideas, and with these views in mind he developed the procedure for the production of his antigen described in the following paragraph.

Mass cultures of bacteria are grown, every effort being made to maintain them in highly antigenic phase. The cells are washed free of metabolites in isotonic Locke's solution. The washed cells are then placed in a special ball mill, and after a certain number of hours, are mechanically disrupted. The intact cells are removed by ultrafiltration. The filter permits the passage of substances in solution and cell fragments, but no cells. The filtrate is then standardized on the basis of native protein content, and the antigen is ready for use.

The nitrogen content is very low, and it is hardly possible to develop a foreign protein, or allergic reaction with the use of the UBA. For this reason in allergic individuals with acute upper respiratory infections the UBA may be given with implicit confidence that no untoward reaction will result.

The undenatured antigen is given intra- and subdermally. The initial dose is 0.1 cc. intradermally, and 0.1 cc. subdermally. If no reaction results, the next dose is the same in the skin, but under the skin it is doubled; that is, 0.2 cc. is given. At the next injection the skin dose is the same and the subcutaneous dose is raised to 0.3 cc. Continuing thus, the skin dose is maintained at 0.1 cc. of the antigen; the subcutaneous dose is gradually increased from the 0.1 cc. dosage up to 0.4 cc. I have not found it necessary to exceed the larger dosage. In other words, no more than 0.5 cc. was used at one dose. Good results are obtained with the small dosages and nothing is to be gained by producing a reaction in the patient. Krueger advises against a maximum dosage, producing a reaction, and in my series of cases I have found it unnecessary to utilize the larger dosage. Injections are given daily or every other day in the acute cases for one or two or three doses, then injections are given three times a week. I have utilized one needle puncture for the two injections, giving first the intracutaneous 0.1 cc. with a tuberculin syringe, then pushing the needle further under the lower layer of the skin and giving the subcutaneous dose with the same puncture. Kracaw in his injections uses two different punctures at a space from each other. I do not believe it is necessary to use the double

puncture, and I think the patient should appreciate the single puncture method.

I have observed the use of the UBA over a year now, and my series of cases involve all seasons of the year, therefore it is probable to expect that the varying organisms which occur in this locality have been met with; and the results speak for themselves.

I believe there is a certain amount of specificity of the organisms exhibited, because the results were usually very excellent or in some cases none at all. This I can explain only on the basis of specificity of organisms, and the cases in which no results were obtained were of such a different strain of organisms from the antigen injection, that the therapeutic value was nil. Likewise in the cases where the results were marked as to practically cure the patient in 24 hours with one injection, we must admit, that the specificity is likewise very high.

Most of my cases were acute upper respiratory infections, and they were judged fairly, and with as cold reasoning as possible. A series of 75 cases, accurately observed and recorded, are included in this report. Detailed case histories are not included, although they are written and in the files.

During the entire injections at no time was any allergic phenomena observed, although it was carefully looked for. There was only one marked reaction, and I believe it is accounted for on the basis of some vascular trauma or injecting into a vein. This severe reaction was not followed by abscess formation or any very pronounced symptom, but was painful for a period of two days locally. Injections are practically painless, and very few patients complain of them, however, since 0.1 cc. is given intradermally naturally the layers of the skin are separated, and the discolored area resulting persists for much longer periods of time than with subcutaneous injections. Where this has bothered patient injections have been given in other areas where they would not be so noticeable.

Dr. Krueger also makes a bacterial antigen for topical application, but none was used in this series of cases, as it was thought a proper evaluation of the value of the injection

could be obtained if only one method was used at a time. For that reason this series includes only the UBA injections, and no topical applications.

It was found that in an acute upper respiratory infection either in the nose or throat, the initial injection of 0.1 cc. intradermally, and 0.1 cc. subcutaneously, usually gave a marked beneficial effect in 24 hours. This was often obtained when nothing else was done except the injection. That is no local treatment was given. In the case of a throat infection, the throat was not painted, in the case of a nasal infection no nose treatment was given. In one case X-rays were made to check up on the sinus condition before and after treatment, and no local treatment whatsoever was used. Clearing of the sinuses is definitely demonstrated. I feel that when the UBA alone is given in this manner, and the patient shows a marked improvement the next day, definitely we might attribute this improvement to the medication. In these cases, however, it was found that if the patient received his initial injection, and got complete relief; unless a prophylactic series of at least two weeks with three injections a week were instituted, that in about 10 days the patient would return with the same symptoms, and the same clinical picture as the initial infection. Following this sad occurrence in a number of cases it was made a routine procedure to never give one injection and quit, but to follow this up with injections three times a week for at least a period of two weeks. Following this procedure the relapse was uniformly avoided, and patient remained well.

In some acute cases there was seemingly no benefit whatsoever from the injections. These cases however were relatively few, and constituted about 4 per cent of my cases. I explain this lack of improvement to the specificity of the organisms, as I have said before. Kracaw reported a series of 45 cases of chronic sinus infections treated with the antigen, and in these cases he used an autogenous organism to make the antigen. Naturally I did not have the facilities for this work, and so can only surmise that the lack of improvement in 4 per cent of these cases is due to different strains of organisms.

The results are tabulated as excellent, good, fair and none. An excellent therapeutic result was one in which improve-

ment was very marked in 24 hours, and patient was practically well the next day. A good result was one in which improvement was marked in two or three days. A fair result is one in which improvement was marked in five days. No result was one in which the infection did not seem to be influenced by the antigen injection. The same classification for 20 chronic cases is used as for the acute cases. Naturally in the chronic cases there were no excellent results, in the sense of a cure in 24 hours.

In the acute cases 40 per cent showed excellent results, in other words the patient was practically cured the day following the injection. Thirty-one per cent showed good results; that is, the patient was cured in two or three days; and 25 per cent showed fair results, in other words, patient was well in five days. As stated before, 4 per cent showed no improvement.

In the chronic cases there were no excellent results naturally. Twenty-five per cent showed good results; that is, patient was well in two or three days. Twenty per cent showed fair results; that is, patient was well within a week; and 55 per cent showed no results. Kracaw in his series of 45 chronic cases reports 95 per cent are cured, following injections over a period of two months. I have not been as fortunate with my chronic cases. From this it would seem that it is an excellent therapeutic agent, in acute conditions. I have so found it clinically.

In the chronic cases I have found that treatment must be instituted over a prolonged period of time. In some instances six and eight months. Some patients would clear up from draining pus with the injections in a month or two, and following the discontinuance of injections, or else the slowing down of injections, the pus would return to the nose, and patient would be complaining of the same symptoms as before treatment was instituted. This would seem that the immunity was rather fleeting. Usually following institution of the three injections a week the chronic cases would clear up again, and remain free as long as the injections were given.

Prophylactically the three injections a week seemed to protect the patient from acute upper respiratory infections.

Approximately 10 cases of furunculosis of the external ear canal were given injections, with practically uniform cures in

each case. These are not included in the cases of upper respiratory infections reported in this paper, and are merely mentioned in passing.

In some cases, particularly the chronic cases, no results were obtained with the 0.5 cc. dosage, but the usual results were obtained when this dosage was increased 0.2 cc. or 0.3 cc.

SUMMARY AND CONCLUSION.

UBA (Krueger) is a therapeutic agent of value in treating upper respiratory infections, giving almost overnight relief in practically half the cases. Fifty-three acute cases were treated with this antigen, with the results shown in the table.

Fifty-three Acute Cases			
Excellent	Good	Fair	None
24 hour results	48-72 hour	120 hour	
40 per cent	31 per cent	25 per cent	4 per cent

The material used was furnished by Eli Lilly and Company of Indianapolis, Indiana.

808 Maison Blanche Building.

**A NEW SYRINGE FOR REMOVING CERUMINOUS
IMPACTIONS FROM THE EXTERNAL
AUDITORY CANAL.**

DR. L. E. HETRICK, New York.

In presenting this instrument, the author has in mind what to him have been disadvantages in the bulky Pomeroy and similar ear syringes; namely their weight, their large size and the large size of the stream issuing from the nozzle. The syringe herewith presented has a 10 cc. barrel with a slender nozzle bending from the main shaft of the tip at an angle of about forty-five degrees. The syringe is decidedly smaller than the Pomeroy, decidedly lighter and easier to handle and the stream has a diameter of approximately 1 mm. The tip should be inserted into the external auditory canal paralleling and in contact with the superior wall. The small stream passes by



the impaction, impinges upon the drum membrane and is reflected outward so that its force is directed against the inner surface of the impaction, producing a *vis a tergo* from within outward. This syringe has the added advantages of permitting the operator to see exactly what he is doing, it is not uncomfortable to the patient and there is relatively no back spatter so that a shield such as is used on the Pomeroy syringe is unnecessary. After several years of successful use of this syringe, the author, at the suggestion of several of his friends who have been using the syringe, presents it to the profession and feels that it will meet a long felt want in a simple but sometimes trying procedure.

40 East 61st Street.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Dec. 15, 1934.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTOLARYNGOLOGY.

Meeting of Dec. 19, 1934.

A. Safe and Humane Method of Anesthesia for Tonsil and Adenoid Operations in Young Children. Dr. James T. Gwathmey.

(To be published in a subsequent issue of THE LARYNGOSCOPE.)

Etiology of Retrobulbar Neuritis. Dr. John H. Dunnington.

(To be published in a subsequent issue of THE LARYNGOSCOPE.)

DISCUSSION.

DR. THOMAS H. JOHNSON: Retrobulbar neuritis, not caused by alcohol, tobacco, lead and various other chemical agents, has been the symptom of a more serious neurological malady in most of the cases that I have seen.

De Schweinitz, Holmes and others have stressed the importance of scotomas in the visual fields as an early sign of tumors near the chiasm. de Schweinitz believes that the optic neuritis in such cases is due to toxins thrown off by the tumor cells penetrating the optic nerve sheath and acting upon the more delicate fibres of the optic nerves. As cerebral neoplasms may grow very slowly the retrobulbar neuritis may precede the manifest presence of the tumor by a period of years.

Practically all modern writers upon the subject have recognized the importance of central scotomas as an early sign in frontal lobe tumors of the brain. Kennedy has stressed the importance of this symptom, and Lilly has reported a definite central scotoma in seven of 14 cases of verified frontal neoplasms. I have found central and paracentral scotomas a common early sign in basal frontal lobe tumors. The following cases illustrate this condition:

Case 1: W. K., a boy, age 11 years, was admitted to the Neurological Institute on Sept. 15, 1933. He gave the following history: In August, 1931, while playing with his companions he lost the sight of the right eye suddenly. He consulted an oculist, who had the sinuses X-rayed. Notwithstanding normal appearing sinuses clinically and by X-ray, the sinuses were opened. Gradually he became totally blind in the right eye and the vision in the left eye had become impaired.

He was found to have a right optic atrophy with a papilloedema in the left eye. The right was blind and there was an absolute central scotoma in the left visual field. On Sept. 19 Dr. Deery operated and found a large angioma beneath both frontal lobes of the brain.

The sudden loss of vision in the right eye was probably due to a hemorrhage from a growth that was already making pressure upon the right optic nerve.

This patient had gone nearly three years from his first symptoms before a diagnosis of brain tumor was made.

A second illustrative case was: R. L., a boy, age 16 years, whose vision had been failing for two years. He had had headache and a pain in the eyes for nine months. When seen at the Neurological Institute on Nov. 4, 1932, he had a very advanced papilloedema and his visual fields showed a large absolute

cecocentral scotoma in the right eye connecting with a large superior hemianopic defect, and an absolute cecocentral scotoma in the left eye. The patient died and at necropsy a glioma of the third ventricle was found.

Here again was a patient who had had failing vision for two years and a diagnosis of brain tumor had not been made until he was in a desperate condition.

Numerous cases have been reported in the literature of disseminated myelitis, a premonitory symptom of which has been a retrobulbar neuritis coming on several weeks before the active manifestations of the true condition have presented themselves.

Such was the case of P. W., a boy, age 15 years. He was seen by me on Aug. 25, 1927. While in camp, two weeks before, the vision of the right eye had become blurred. When I saw him his vision in the O.D. was 20/20+ with correction, the O.S. nil. X-ray of the sinuses was negative. Two days later the vision in the O.D. had been reduced to finger count at 2 ft.

Acting upon the advice of Dr. Foster Kennedy, who had made a neurological examination of the patient, the late Dr. Thomas Atkins operated and reported that he had found no pathology of any kind in the ethmoid or sphenoid cells. About two weeks after the sinus operation a weakness of the lower extremities developed. This was followed in the course of a few days by a paralysis of the legs, arms and abdominal muscles. The patient eventually died from a disseminated myelitis.

I may say here that some neurological authorities believe that disseminated myelitis is a form of acute multiple sclerosis.

In my work at the Neurological Institute I have seen a considerable number of cases of multiple sclerosis and one of the common early symptoms is a transient reduction in the visual acuity and the presence of a scotoma that may be temporary or permanent. Henson has stated that the central scotoma of multiple sclerosis is never longer than three months' duration. The visual symptoms may antedate the severer neurological changes by months and even years.

Such transient attacks of retrobulbar neuritis are not confined to multiple sclerosis but occur in other organic diseases of the central nervous system.

The following two cases have recently come under my observation:

M. D., age 50 years. Seen at the Neurological Institute on Dec. 14, 1934. He had been referred for a neurological examination from the Manhattan Eye and Ear Hospital with the following report: The patient was seen in the outpatient department on Nov. 20, 1934. Ten days previously he had had a severe cold with fever and headache, and five days later he lost the vision in the right eye. Upon examination it was found that he had an acute optic neuritis in the right eye and the vision was finger count at 2 ft. There was an extensive central scotoma to form and colors in the right eye. The Kahn and blood Wassermann tests were negative. X-ray report: Moderate involvement of both ethmoids with considerable absorption and osteosis, right antrum cloudy, sella normal. The left eye was normal. No operation had been done upon the sinuses. I saw the patient on December 14, 1934. The corrected vision in the right eye was 20/30 -1. The right pupil was smaller than the left and reacted very slightly to light. The right disc margin was slightly blurred and the right nerve head slightly pale. The left eye was normal. A fine nystagmus was developed in the lateral fields of gaze. A neurological examination showed a generalized tremor of the body, a bilateral Hoffman, a definite right Babinski with a suggestive left Babinski. This case has had a return of vision from finger count at 2 ft. to 20/30— in less than a month without treatment. He now has definite signs of an as yet undiagnosed disease of the central nervous system.

F. M., male, age 59 years. About the middle of September vision became blurred in both eyes. The patient saw Dr. Angus MacLean on Sept. 21, 1934, who found a central scotoma for colors in both eyes. The peripheral fields were normal. The corrected vision was 20/30 in the right and 20/40 in the left eye. A thorough physical examination was negative.

I saw the patient on Nov. 22 in consultation. At that time he had, with correction in the right eye 20/15, and in the left eye 20/20+ vision. The pupils reacted slightly to light and there was a paracentral scotoma in each eye. There was a left facial weakness and the patient stated that recently he had had some trouble in swallowing, had frequently bitten the right side of his tongue and that he had some urgency in urinating. Memory was impaired for recent events.

Within a period of three months, without treatment, the central scotomas for colors have disappeared, the vision has returned to normal, but there is a paracentral scotoma, homonymous in type in each visual field, and he has definite signs of a lesion of the brain involving the visual pathway and other structures in the right hemisphere.

At times no cause can be found as in the case of H. B., a college student who was admitted to the Neurological Institute, March 5, 1930. For two months the vision in the right eye had been blurred. When I saw him the vision in the right eye was 20/20—, in the left eye finger count at 10 in. The visual field of the right eye was normal. There was an absolute central scotoma in the left visual field.

A thorough work up in the hospital revealed no cause for the visual disturbance.

After two weeks of rest in the hospital without treatment of any kind the scotoma disappeared, the vision returned to normal, but a slight pallor of the left optic disc developed. I have followed this case and when seen recently there had been no new developments.

It seems to me that since Sluder popularized sinus surgery in diseases of the optic nerve, the role of sinusitis in retrobulbar neuritis has been overemphasized. Sinusitis, varying from mild infections to purulent pansinusitis, is a very prevalent condition. Yet, how often do you see retrobulbar neuritis? I have been forcibly impressed by the large number of cases of brain tumor that I have seen at the Neurological Institute that have had radical sinus surgery done for the relief of the visual disturbances which have been the earliest symptoms of the cerebral neoplasm. Walker found sinusitis to be the cause of retrobulbar neuritis in only 4 per cent of his cases.

I believe that cases of optic neuritis of all sorts should have careful and thorough neurological studies made, whether the sinuses are believed to be the cause or whether or not sinus surgery has been done.

The Present Status of the Submucous and Turbinate Operation. Dr. Westley M. Hunt.

(To be published in a subsequent issue of THE LARYNGOSCOPE.)

Present Status of Mastoidectomy. Dr. Wesley C. Bowers.

(To be published in a subsequent issue of THE LARYNGOSCOPE.)

DISCUSSION.

DR. TRUMAN L. SAUNDERS: I am in complete accord with the principles so ably enunciated by Dr. Bowers. I cannot disagree with any of them, and I shall confine my brief discussion to further emphasis of certain points which I deem of interest.

In the first place, the objective of the simple mastoid operation is a dry ear with good hearing and a well healed mastoid wound. This is what the laity can reasonably expect from the aural surgeon if he has had the case under his care from the beginning of the ear disease, and it well behooves that individual, if the case deviates from this ideal in any particular, to have good and sufficient reasons why the defect occurs.

In regard to the question of disease of the petrous apex, perhaps that does not quite come under the scope of the simple mastoid operation, but I believe as we go on in the study of this disorder that we will classify our cases more and more into those that are favorable for operation and those that are not. Furthermore, I think it is necessary that we study the influence of a well performed simple mastoid operation on the prevention of this unpleasant complication.

I wish to say a word about the mastoid dressing and the healing of the mastoid wound. I think quick healing of the mastoid wound is a surgical stunt which at times jeopardizes the hearing of the patient. I firmly believe that the primary object of the mastoid operation is to restore the hearing in the middle ear and to heal the mastoid.

Now, in regard to the time for operating, I again wish to emphasize Dr. Bowers' point and to simply remind you that an early operation makes neither for early healing or absence of complications. In my personal experience I have operated on a case within 48 hours on account of an increasing toxic acidosis as well as more severe mastoid symptoms, but it was the acidosis that made the early operating imperative. That case went on to sinus thrombosis. In that one particular instance early operation did not prevent the complication. I wish to say also a word about too much conservatism in waiting for operation. It is no credit to an aural surgeon to carry a patient along without an operation and at the end of six or eight weeks have him with a dry ear but with an ear that is deaf and a deafness that is accompanied by a high-pitched tinnitus. These cases remain deaf and the tinnitus usually persists for the life of the individual.

In closing, I would beg to remind you that in these mastoid cases that are left without operation very often there is an area of weakened resistance in the mastoid and the next severe nasopharyngeal infection will precipitate an acute attack, which, as Dr. Bowers has stated, must be operated at once. Such an attack is much more liable to complications than the primary condition. Furthermore, I would like to call attention to the fact that many cases of recurring otitis are nothing but flare-ups of a focus in the mastoid process. Every case of persistent recurrent acute otitis in which the nasopharynx has been examined and sinus disease and tonsils and adenoids have been excluded should always have mastoid X-rays. These cases are very frequently caused by a focus in the mastoid process.

DR. DAVID H. JONES: I want to assure you at the outset that there is nothing personal in my remarks about Dr. Gwathmey's paper. I know him very well and we have talked this matter over, but I do not think it is proper for a paper like that to go out from the Academy without a protest. Dr. Gwathmey has told us how nicely the child goes under but he does not tell us how long the child stays under. That, to my mind, is very important. In following the work of Dr. Myerson and Dr. Jackson, who investigated these cases after tonsillectomy, we find blood in the trachea. According to Dr. Jackson's dictum the cough reflex is the watchdog of the lung, and anything, either local or general anesthetic, that prolongs the absence of that reflex is dangerous for our patient. I happened to read Dr. Coburn's paper in regard to the work done at the Woman's Hospital, in which he reported 654 operations done in 1932. All the patients were treated by the administration of carbon dioxide (10 per cent carbon dioxide and 90 per cent oxygen). Their pulmonary complications were 0.31 per cent. Thinking it might be an excellent idea to try out at the Manhattan, we tried it out in one operating room. We have two tonsil

wards. The nurses did not know we were trying this out. After a few days these same nurses, who have been there a number of years, asked why it was that some of these patients came out so quickly. They noticed that they began to fight immediately upon coming back to the wards. We said nothing at all but kept on trying it. At first we tried it only on 10 patients. Now we are using this method in both operating rooms. To date we have operated on over 10,000 patients, with our pulmonary complications almost *nil*. Of course, you will want to know the cost. The cost now for each patient is about 5 cents.

DR. RAYMOND C. COBURN: I am very much interested in the report by Dr. Jones of the administration of carbon dioxide after operation at the Manhattan. I have been an advocate of the postoperative administration of carbon dioxide to deoanesthetize patients for 15 years. Early in 1920 Prof. Henderson of Yale; Dr. Haggard, his assistant, and your humble servant made a report after an investigation of over two years with the administration postoperatively of carbon dioxide to deoanesthetize the patient and to establish the physiological effects of plain carbon dioxide upon human subjects. This was as we understand it, the first application of plain carbon dioxide to the human subject. We found that the effect of carbon dioxide was very beneficial in several ways at that time. The patient recovered from the anesthetic much more quickly. The blood carbon dioxide content was returned to more nearly the normal preoperative Ph concentration. The blood pressure also had a tendency to be brought to its preoperative condition.

About five years ago the postulation of the theory that the early postoperative pulmonary complications were caused by respiratory depression of the anesthetic gave a great impetus to the use postoperatively of carbon dioxide. It was in that connection that at the Woman's Hospital, from the years 1930 to 1932, inclusive, 1837 consecutive gynecological laparotomies were reported, with only six postoperative pulmonary complications. This makes it less than one-third of 1 per cent. The reason this report was made was that Dr. King, reporting from the Massachusetts General Hospital in Boston on the same type of operation, reported on 148 gynecological laparotomies with practically 30 times that percentage of pulmonary complications. In his cases carbon dioxide was not used.

The chief objection to the use of the nonvolatile anesthetics is that you cannot pump out the anesthetic by the increase of ventilation with administration of carbon dioxide.

In tonsillectomies and in most of the operations done by this Section you already have in the patients' upper respiratory tracts an increased number of pathogenic germs. The trauma of the operation, plus the irritation of the anesthetic, increases the production of mucus, and this mucus enmeshes the pathogenic bacteria. Postoperatively the drainage is naturally toward the lungs. Prevention requires an active reflex of the larynx.

DR. JACOB BRAUN: Safety in operations for tonsils and adenoids in children cannot be attained without a good anesthesia. The mode of procedure in this part of the operation plays a highly important part in obviating the troublesome and occasionally tragic complications which may follow.

For many years I have stressed the necessity of observing every safeguard throughout narcosis—before, during and after completion of the operative procedures, until the return to complete consciousness.

Our method has been as follows: Ethyl chloride by open mask drop, gradually administered, initiates the anesthesia and is continued until the exact point of muscular relaxation and smooth diaphragmatic breathing is reached. At this stage the anesthesia is continued with ether mask and surgical narcosis established, maintained throughout the operation with ether vapor. The abolishment of the pharyngeal reflex facilitates greatly the necessary manipulations, diminishes operative trauma and, most important, obviates the aspiration into the tracheobronchial tree of secretions, blood and detritus.

The operation completed and all bleeding controlled, the surgical field remains under close visual observation under full illumination until return to full consciousness. As the pharyngeal reaction returns the gagging and vomiting serve to empty the stomach and pharynx of all secretions, and these are immediately aspirated from the pharynx with the suction tube. The cough reflex incident to this stage serves to free the larynx of any secretions that may have accumulated. It is in this stage that any bleeding that may start as a result of the violent muscular efforts can be controlled, often by a few seconds of further administration of ether vapor, until muscular reaction is diminished, or by the application of a tie.

The return to consciousness having been watched, and every safeguard taken, the patient is returned to his room, lying flat on his stomach, and face toward the left. Any secretions will flow freely from the mouth without danger of aspiration.

Since we have followed this procedure, not only have we been remarkably free from complications, but the shortened and more pleasant convalescence has been most marked.

DR. ARTHUR J. HERZIG: I am thoroughly in accord with everything that Dr. Bowers said in regard to the simple mastoid operation. I would just like to add one or two things which I think might be of interest.

One is the use of catgut sutures for suturing the periosteum first. I then suture the skin or use Michel clamps or silkworm gut. The second point is the use of acriviolet in 1:200 solution for all mastoid wounds and dressings. The dressings are saturated with this solution. It stimulates granulation and eliminates odors around the wound, and keeps the wound dry and clean. Granulation time is much less. I have had three cases of mastoidectomy in which I have used this solution and in which I have sutured the periosteum and closed the wound completely. They were completely healed in four to seven days. I have watched these cases for weeks and there was no recurrence of discharge.

DR. WILLIAM WESLEY CARTER: If I did not have absolute confidence in the friendship of the members of this Section, many of whom I have been associated with for 35 years, I would not express this little item, in reference to the submucous operation. I devised that operation myself while working as clinical assistant in Dr. Charles H. Knight's Clinic in the old Manhattan Eye and Ear Hospital. I have been deeply interested in the operation ever since and devised the first instruments that were used in performing it. I did not get credit for it. The credit was carried 3500 miles away from New York to another man.

There are some things that owe their chief value to their antiquity. I hope you will not consider this as having only that virtue. This was written by me in 1906 and published in *THE LARYNGOSCOPE* in June, 1906:

"While I am convinced that the septum as a vertical support takes no part in the preservation of the contour of the nose, I am equally certain that it does play a most important part as one of the forces in the development of the symmetrical adult organ and therefore should not be removed during the years of most active growth."

That opinion was expressed at that time and it is true today. During the passing years I have seen a great many cases of nasal deformity that have been due to the submucous operation, where the upper portion of the cartilaginous septum has been removed from its proper place between the lateral cartilages. This has been done in many instances by the use of the Ballinger swivel knife, which causes traction on the cartilage as it is drawn out and pulls it from its proper position.

I do not believe that the submucous operation should be done under 12 years of age. Dr. Hunt has referred to my views upon this subject and to my modified submucous operation to be used in the case of septal deviations in children.

I use only three instruments in performing the submucous operation. These were the original instruments, the punch forceps, the submucous elevator and the small knife which is used for that purpose, and the special speculum. It is my belief that a multiplicity of instruments is confusing to the operator and does not add to his efficiency.

There was one point which was not mentioned by Dr. Hunt, and that is in reference to the reproduction of the septum. The septum is not regenerated after its removal. The reason for that is that the septum is formed in membrane, and bones and cartilage that are formed in membrane are never reproduced after they have been removed because the osteogenetic centre is destroyed when you take out the bulk of the septum.

DR. CAESAR HIRSCH: I would like to add a very instructive example to the very interesting paper of Dr. Dunnington. It was in the year 1920 that an ophthalmologist sent a 21-year-old girl to me with a retrobulbar neuritis on the left side. I examined the nose and sinuses and I could not find any pathology, and reported this to him. The girl, who happened to be the daughter of a very rich man, traveled all over Germany seeing ophthalmologists and seeing neurologists. No one could find any reason for this left-sided retrobulbar neuritis. So, perhaps after five months, the girl came back to me and upon the urgent insistence of the ophthalmologist and neurologist I performed an ethmoidectomy and sphenoidectomy without being able to find any pathology; however, two weeks after the operation her condition cleared up and the retrobulbar neuritis disappeared completely. I was blamed very much by the ophthalmologist and neurologist because I had been so stubborn and had not operated on this case some months earlier; but, after half a year, this girl developed multiple sclerosis, and since that time she has developed the worst case of multiple sclerosis that I have ever seen. This case, therefore, seems to me very instructive for us as rhinologists. It teaches us not to operate on such cases if we have no reason in our field to interfere. This girl had a remission of her beginning multiple sclerosis such as we see very often. Coincidentally it happened that this remission took place 14 days after the sinus operation, which of course had nothing to do with it.

At the same time Herzog, then otologist in Innsbruck, reported some cases of retrobulbar neuritis before the German Oto-Laryngological Society, in which he had operated on the sphenoid and the ethmoid with very good results. He explained that on the finding of some little edematous changes in the mucous membranes of the sinuses. I do not know whether some of his cases also happened to be early cases of multiple sclerosis.

In regard to the paper by Dr. Bowers I would like to say, and perhaps it depends on my poor command of the English language, that I do not understand why this kind of mastoid operation is called "simple." I have the idea that these "simple" mastoids are very "severe" mastoids insofar as we should in my opinion be very radical in this operation. If we have made up our minds to operate in these cases we should operate them in a very radical way, just as in the so-called "radical" mastoid operation we are operating now in a very conservative way. I think that in these "simple" mastoids we should clear up all the cells so that at the end of the operation we have made a very good specimen in which no pathological cells can be found. I feel that if we would operate on these cases in this manner, perhaps the number of complications would be very much less.

DR. WESLEY C. BOWERS: In regard to sewing the periosteum in a mastoid operation I find that I cannot do it. I cannot get the periosteum together. I do the best I can along with the subcutaneous tissues. I think you will find that there is quite a little space between the edges of the periosteum.

In regard to healing in one week, I still think that the object of the mastoid operation is to remove disease and create drainage. There are a lot of cells which we do not reach no matter how much we know about it. We cannot get some of the cells beyond the labyrinth and in the middle ear, and we must drain these cells or be forced to do a radical mastoidectomy later. Disease in these cells is accompanied by persistent drainage.

I am very glad that Dr. Hirsch mentioned the point about simple mastoidectomy. I think that word should be changed to some other term, such as complete mastoidectomy. It is very deceiving. Of course, when you do a simple mastoid operation, you do it radically, cleaning out every cell you can possibly reach.

DR. JAMES T. GWATHMEY: It was most unfortunate that the lengthy program precluded the satisfactory reading of my paper. Hence, Dr. Jones, Dr. Coburn and Dr. Braun did not discuss my paper at all, but merely gave their opinions about subjects that were entirely foreign to the paper. None of them seemed to understand the value of preliminary medication in excluding pathological lesions of the lungs.

The loud stertorous breathing that is usual with the methods now in general use is more apt to suck blood into the trachea than a more quiet induction with even breathing throughout the operation. The cough reflex returns with this method much quicker than with any method now in use, except straight nitrous oxide and oxygen. Preliminary medication is just as important as carbon dioxide and oxygen at the completion of the operation. In fact, with a quick operator, the cough reflex returns without the administration of carbon dioxide and oxygen at all. The patient is never saturated with ether. Consequently we do not have the fighting, gagging, coughing, and delirium that usually follows the old methods of administration.

Nitrous oxide and oxygen costs more than air, but the patient is the gainer by this change. When you consider the cost of the ether, oxygen, carbon dioxide, the salary of the resident anesthetist, light, etc., the statement of Dr. Jones that the cost for each patient is 5 cents is too silly to discuss. Dr. Braun would be disappointed if he used this technique, as he would miss the gagging and vomiting on the table. I agree with Dr. Coburn that nonvolatile anesthetics can not be pumped out by an increase of ventilation, but no nonvolatile anesthetics are used with the method as outlined. The amount of nembutal used produces only a quiet sleep. The irritation of the anesthetic, as suggested by Dr. Coburn, and the mucus are absent. We have an active reflex of the larynx immediately after the operation and it is no more possible for the child to swallow blood after operation than it is for a patient in natural sleep.

This longer postoperative sleep by keeping the throat reflexes quiet and by not disturbing the clots formed, is unquestionably a factor in preventing postoperative hemorrhage even if ties have been used. Sington, using paraldehyde per rectum, produces a sleep preoperatively and also a postoperative sleep of several hours.

The procedure as outlined is simpler and easier of execution, especially in a large clinic. If any fault can be found with this method, it is that the child wakes up too soon. The clinic is the final test of any and everything in medicine and surgery. We believe that a distinct advance has been made in having the child soundly asleep before operation.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

Meeting of Jan. 17, 1935.

Report of Endoscopic Work Extending from June, 1933 to January, 1935. Dr. Guy Maness.

Esophageal foreign bodies (26 cases): Meat, 4 cases. Evaporated peach, 1 case. Coins, 7 cases (3 pennies, 3 nickels and one 50-cent piece). Bones, 5 cases (2 chicken, 2 beef and 1 fish). Cartilage, 1 case. Screw (large), 1 case. Button (large), 1 case. Safety pins, 2 cases. Cockle burr, 1 case. Dental plate, 1 case. End of tin whistle, 1 case. End of adhesive tape spool, 1 case.

Foreign bodies in larynx (6 cases): Straight pins, 4 cases. Safety pins, 1 case. Needle, 1 case.

Foreign bodies in trachea and bronchi (20 cases): Grain of corn, 4 cases. Peanut kernels, 2 cases. Peanut shells, 2 cases. Black-eyed pea, 1 case. Bean, 1 case. Watermelon seed, 2 cases. Cedar wood, 1 case. Cartilage, 1 case. Nails, 3 cases. Carpet tack, 1 case. Tin off of lead pencil, 1 case.

Five esophageal carcinomas were diagnosed by esophagoscopy and biopsy. Eight esophageal strictures have been treated. Seven were lye strictures and one was luetic. Five cases of cardiospasm were treated. One esophageal diverticulum was found in a case of suspected foreign body. One infant, age 10 days, with congenital atresia of the esophagus was esophagoscoped. About 10 or 15 cases of suspected foreign bodies were "scoped" and nothing found. Two cases of multiple papillomata in children were treated. Five cases of carcinoma of larynx. Four cases of benign laryngeal tumors were removed. Three cases of laryngeal stricture were treated. One case of tracheal stenosis was treated. One tracheal polyp was removed. Three cases of bronchogenic carcinomas were found.

A considerable number of diagnostic laryngoscopies were done, the exact number of which I have no record.

A number of diagnostic and therapeutic bronchoscopies were done on patients with lung abscess, bronchiectasis, asthmatics, obscure pulmonary bleeding, suspected lung tumors and cases of atelectasis.

Results of Cases of Foreign Body: Recoveries occurred in all but two cases which terminated fatally. One fatal case was that of a boy, age 3 years, who had a peanut kernel in the bronchus. He was bronchoscoped without anesthesia and the peanut removed without much difficulty. Immediately after the bronchoscope was removed the child ceased to breathe. He made absolutely no attempt to breathe. All efforts of resuscitation failed. The death was not definitely accounted for. The most likely thing seemed to be due to pleural shock.

The other fatal case was that of a man, age 61 years, who had a sharp-pointed bone in the esophagus for 11 days. Attempts to push the bone into the stomach by blind methods had been done by his doctor. When first seen the

patient was acutely ill and had X-ray evidence of a marked mediastinitis. An esophagoscopy was done and the bone removed. It was found that the esophagus had perforated and a large amount of extremely foul smelling pus was evacuated.

An operation for drainage of the mediastinum was done by Dr. Blalock, but the patient died in less than 24 hours. This patient obviously was hopeless when first seen and illustrates the danger of trying to push sharp pointed foreign bodies into the stomach by blind procedures.

One patient, a man, age 56 years, had an extremely large bone in the upper end of the esophagus. The bone was so large that attempts at removal failed. An incision was made externally and the bone removed. The patient recovered after a rather stormy convalescence.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

Meeting of March 15, 1935.

Brain Abscess. Dr. Herschel Ezell.

E. C., age 12 years, was brought in Nov. 1, 1934, complaining of pain over the right eye, nausea, vomiting and emaciation.

History of present attack: The patient has been suffering from the symptoms as related above for five weeks. Most of this time she had fever ranging from 100° on most occasions to 104° at times. The child gave a history of having lost a great deal, but an undetermined amount, of weight as she had not been weighed for some time. The left ear, previously operated on, had discharged about twice annually following contraction of colds. She had been treated for malaria, receiving large doses of quinine without benefit. Finally, this line of treatment was abandoned and she was referred to me on suspicion of ear complications.

Past History: I operated upon this patient for acute mastoiditis of the left side on March 26, 1925. Referring to old records I find that mastoiditis was of three weeks' duration before operation and that the child gave no history of pain.

A simple mastoidectomy was done at St. Thomas Hospital and the patient made an uneventful recovery. About 10 weeks later, or on June 8, 1925, I removed her tonsils and adenoid. After a normal postoperative convalescence she was brought to me on Nov. 1, 1934.

Examination: The patient was found to be quite emaciated, pale and anemic. The right ear was negative. There was a small perforation of the left drum at its upper posterior portion and the ear was discharging slightly. The throat was negative. The mucous membrane of nose was inflamed and there was some scabbling near its orifices, but no pus was found. The right side of the septum was bleeding slightly which, together with the induration of the orifices of the nose, was probably due to the patient's persistent scratching and picking at her nose. The pupils were normal in size and reacted both to light and accommodation. There was no interference with the IIIrd nerve and there were normal excursions of the eyes in all directions. The fundus was normal, there being no evidence of papillitis. The patient was considerably nauseated and vomited frequently while I was examining her at 11 A. M., the date of her arrival at the hospital. The usual laboratory work was ordered, such as urinalysis, differential blood count, etc. X-rays of ears and sinuses showed the sinuses negative and a small area of necrosis of the right mastoid. The left one was reported to be a normal postoperative mastoid. Dr. O. N. Bryan was called in consultation in the afternoon of the day of her arrival; he advised a Widal test, a test for tularemia and a blood culture in addition to the laboratory work already ordered. It was apparent at this time that the patient was suffering from some brain involvement, probably abscess.

A spinal puncture was done on Nov. 1 and 4. The fluid was cloudy. The laboratory report on Nov. 1 showed that there was a cell count of 550,000 and on Nov. 4, 570,000. There were no bacteria in either specimen and cultures of both were negative. On Nov. 6, 8 A. M., patient in coma, temperature being 104.4°; pulse, 90; respiration, 50. Child died about 10 A. M.

Summary: 1. This patient probably had an abscess in the left temporo-sphenoid region secondary to middle ear infection. 2. Operation was not done because of absence of localizing symptoms. 3. A postmortem examination was not obtained.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

The Fifty-Seventh Annual Congress of the American Laryngological Association was held at the Royal York Hotel, Toronto, Canada, on May 29-30-31, 1935, under the presidency of Dr. Dunbar Roy. Dr. George M. Coates kindly acted as secretary *pro tempore*.

The following were elected Emeritus Fellows: Dr. Joseph C. Beck and Dr. Clarence C. Rice; and the following were elected to Active Fellowship in the Association: Dr. Curtis C. Eves, Philadelphia, Pa.; Dr. Albert C. Furstenberg, Ann Arbor, Mich.; Dr. Edward A. Looper, Baltimore, Md.; Dr. LeRoy A. Schall, Boston, Mass.

The membership of the Association now stands as follows: Honorary Fellows, 2; Corresponding Fellows, 20; Emeritus Fellows, 19; Active Fellows, 83; and Associate Fellows, 16.

The report of Dr. Berry, Chairman of the Special Committee on "Memorial Fund for the Scientific Advancement of Laryngology — American Laryngological Association," was accepted and a special standing committee was approved by the Association.

The following officers were elected: President, Dr. Burt R. Shurly. First Vice-President, Dr. Edward C. Sewall. Second Vice-President, Dr. H. Marshall Taylor. Secretary, Dr. James A. Babbitt. Treasurer, Dr. C. J. Imperatori. Librarian and Historian, Dr. George M. Coates. First Councilor, Dr. Gordon Berry. Second Councilor, Dr. J. B. Greene. Third Councilor, Dr. Horace Newhart. Fourth Councilor, Dr. Dunbar Roy.

Fellows desiring to present papers at the Fifty-Eighth Annual Congress will please communicate promptly with the Secretary.

Those sponsoring the application of new members will please communicate with Dr. George M. Coates, Chairman of Special Committee on New Members.

In order that prompt notice may be sent to the Fellows, the Secretary would appreciate immediate information on any death in the Association.

